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OF THE

COLLEGE OF PHYSICIANS

OF PHILADELPHIA.

VOLUME THE THIRTEENTH.

TRANSACTIONS

OF THE

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OF

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COLLEGE OF PHYSICIANS OF PHILADELPHIA.

1883.

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LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION.

ELECTED	
1787.	JOHN REDMAN, M.D.
1805.	WILLIAM SHIPPEN, M.D.
1809.	ADAM KUHN, M.D.
1818.	THOMAS PARKE, M.D.
1835.	THOMAS C. JAMES, M.D.*
1835.	THOMAS T. HEWSON, M.D.
1848.	GEORGE B. WOOD, M.D., LL.D.
1879.	W. S. W. RUSCHENBERGER, M.D.
1883.	ALFRED STILLÉ, M.D., LL.D.

* Died four months after his election.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

JULY, 1883.

[Non-Resident Fellows are marked thus (*).]

ELECTED

1870. Adler, John M., M.D.

1859. Agnew, D. Hayes, M.D., LL.D., Professor of Surgery in the University of Pennsylvania, Surgeon to the Pennsylvania Hospital, Consulting Surgeon to the Orthopædic Hospital and to St. Christopher's Hospital, and to the Philadelphia and Northern Dispensaries.

1876. Alison, Robert H., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1867. Allen, Harrison, M.D., Professor of Physiology in the University of Pennsylvania, Consulting Surgeon to the Mission Hospital.

1873. Allis, Oscar H., M.D., Surgeon to the Presbyterian Hospital and to the Howard Hospital.

1869. Andrews, T. Hollingsworth, M.D., Surgeon to the Howard Hospital, Consulting Surgeon to the Hospital of the Good Shepherd, Radnor.

1882. Ashbridge, Richard, Assistant Surgeon U. S. Navy.

1872. Ashbridge, William, M.D., Surgeon for Out-patients to the Pennsylvania Hospital.

1863. Ashhurst, John, Jr., M.D., Professor of Clinical Surgery in the University of Pennsylvania, Surgeon to the Children's Hospital, Consulting Surgeon to St. Christopher's Hospital and to the Hospital of the Good Shepherd, Radnor.

ELECTED

1865. Ashhurst, Samuel, M.D., Surgeon to the Children's Hospital.

1835. Ashmead, William, M.D.

1857. Atlee, Walter F., M.D., Consulting Physician and Surgeon to St. Luke's Hospital, Bethlehem.

1852. Bache, Thomas Hewson, M.D.

1883. Baer, Benjamin F., M.D., Demonstrator of Clinical Gynæcology and Chief of the Dispensary for Diseases of Women in the University of Pennsylvania.

1879. Baker, Washington H., M.D., Obstetrician to the State Hospital for Women and Infants, Assistant Physician to the Philadelphia Lying-in Charity and to St. Christopher's Hospital, Assistant Surgeon to the Orthopædic Hospital, Physician to the Philadelphia Dispensary.

1876. Baldwin, Louis K., M.D., Examining Physician to the Hospital of the Good Shepherd, Radnor.

1880. Bartholow, Roberts, M.D., Professor of Materia Medica and General Therapeutics in the Jefferson Medical College.

1883. Baum, Charles, M.D.

1873. Baxter, H. F., M.D.

1874. Beecher, A. C. W., M.D.

1860. Benner, Henry D., M.D.

1874. Bennett, W. H., M.D., Physician to the Episcopal Hospital and to St. Christopher's Hospital.

*1866. Black, J. J., M.D.

*1867. Boardman, Charles H., M.D.

1859. Boker, Charles S., M.D., Surgeon to St. Joseph's Hospital.

1856. Brinton, John H., M.D., Professor of the Practice of Surgery and Clinical Surgery, in the Jefferson Medical College, Surgeon to St. Joseph's Hospital, and Consulting Surgeon to the Southwestern Hospital of Philadelphia.

1878. Bruen, Edward T., M.D., Assistant Physician to the Hospital of the University of Pennsylvania, and Demonstrator of Clinical Medicine in the same, Physician to the Phila-

ELECTED

delphia Hospital, Examining Physician to the Hospital of the Good Shepherd, Radnor.

1879. Buck, W. Penn, M.D.

*1851. Bullock, William R., M.D.

1870. Burnett, C. H., M.D., Professor for Diseases of the Ear in the Philadelphia Polyclinic, Aurist to the Presbyterian Hospital, Consulting Aurist to Pennsylvania Institution for the Deaf and Dumb.

1880. Chapman, Henry C., M.D., Professor of the Institutes of Medicine and of Medical Jurisprudence in the Jefferson Medical College.

1868. Cheston, D. Murray, M.D., Physician to the Children's Hospital.

1873. Clark, Leonardo S., M.D.

1872. Cleemann, Richard A., M.D.

*1842. Clymer, Meredith, M.D.

1871. Cohen, J. Solis, M.D., Honorary Professor of Laryngology in the Jefferson Medical College, Professor of Diseases of the Throat and Chest in the Philadelphia Polyclinic and College for Graduates in Medicine, and Physician to the German Hospital.

1866. Cruice, R. B., M.D., Surgeon to St. Joseph's Hospital.

1873. Cruice, W. R., M.D.

1868. Cummiskey, James, M.D.

1858. Da Costa, J. M., M.D., Professor of the Principles and Practice of Medicine in the Jefferson Medical College, Physician to the Pennsylvania Hospital, Consulting Physician to the Children's Hospital, to the Northern Dispensary, and to the Pennsylvania Free Dispensary for Skin Diseases.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital.

1874. Deakyne, A. C., M.D.

*1870. Deal, L. J., M.D.

ELECTED

1864. Downs, R. N., M.D., Consulting Physician to the Germantown Hospital.

1864. Duer, E. L., M.D., Accoucheur to the Philadelphia Hospital, Surgeon to the State Hospital for Women and Infants, Visiting Physician to the Preston Retreat.

1871. Duhring, L. A., M.D., Clinical Professor of Skin Diseases in the Hospital of the University of Pennsylvania, Physician to the Dispensary for Skin Diseases.

1881. Dulles, Charles Winslow, M.D., Surgical Registrar to the Hospital of the University of Pennsylvania, Surgeon for Out-patients to the Presbyterian Hospital.

1863. Dunglison, Richard J., M.D.

*1871. Dunglison, Thomas R., M.D.

*1849. Dunott, Justus, M.D.

1860. Dunton, William R., M.D., Consulting Physician to the Germantown Hospital.

1882. Edwards, Joseph F., M.D.

1880. Eskridge, J. T., M.D., Physician to Jefferson Medical College Hospital, Physician to St. Mary's Hospital.

1868. Evans, H. Y., M.D., Physician to the Charity Hospital.

1872. Finn, W. H., M.D., Surgeon to St. Christopher's Hospital.

1866. Fischer, Emil, M.D.

1862. Forbes, William S., M.D., Demonstrator of Anatomy in the Jefferson Medical College, Surgeon to the Episcopal Hospital.

1870. Ford, W. H., M.D.

1864. Fricke, Albert, M.D.

1870. Gardette, E. B., M.D.

*1873. Gerhard, George S., M.D.

1864. Getchell, F. H., M.D., Gynæcologist to the Hospital of the Jefferson Medical College, Obstetric Physician to the Catharine Street Dispensary.

ELECTED

*1846. GIBBONS, HENRY, M.D.

*1848. GIVEN, ROBERT A., M.D.

*1854. GOBRECHT, WILLIAM H., M.D.

1868. GOODELL, WILLIAM, M.D., Professor of Clinical Gynæcology in the University of Pennsylvania, Physician in Charge of the Preston Retreat, Consulting Physician to the Lying-in Department of the Northern Dispensary.

1867. GOODMAN, H. EARNEST, M.D., Surgeon to Wills Hospital and to the Orthopædic Hospital, Consulting Surgeon to the State Hospital for Women and Infants.

1864. GRANGER, WILLIAM H., M.D.

1857. GREEN, ALFRED, M.D.

1870. GRIER, M. J., M.D.

1883. GRIFFITH, J. P. CROZER, M.D.

1842. GRISCOM, JOHN D., M.D.

1883. GROSS, FERDINAND H., M.D., Surgeon to the German Hospital.

1857. GROSS, SAMUEL D., M.D., LL.D., D.C.L. Oxon., LL.D. Cantab., Emeritus Professor of the Principles and Practice of Surgery in the Jefferson Medical College, Consulting Surgeon to the Orthopædic Hospital.

1868. GROSS, SAMUEL W., M.D., Professor of the Principles of Surgery and Clinical Surgery, and Lecturer on Diseases of the Urino-Genital Organs in the Jefferson Medical College and Surgeon to the Hospital of the same, Surgeon to the Philadelphia Hospital, Consulting Surgeon to the Northern Dispensary and to the Pennsylvania Free Dispensary for Skin Diseases.

1871. GROVE, JOHN H., M.D., Surgeon to St. Mary's Hospital.

1863. HALL, A. DOUGLASS, M.D., Surgeon to Wills Hospital.

1865. HAMILTON, GEORGE, M.D.

*1859. HAMMOND, WILLIAM A., M.D.

1865. HARLAN, GEORGE C., M.D., Surgeon to Wills Hospital, Ophthalmic and Aural Surgeon to the Pennsylvania Hospital.

ELECTED

1863. Harlow, Lewis D., M.D.

1862. Harris, Robert P., M.D., Physician to the Franklin Reformatory Home.

1847. Hartshorne, Edward, M.D.

*1851. Hartshorne, Henry, M.D.

*1849. Hastings, John, M.D.

1855. Hatfield, Nathan L., M.D., Consulting Physician to the Northern Dispensary.

1872. Hays, I. Minis, M.D.

1882. Hearn, Joseph, M.D., Surgeon to the Hospital of the Jefferson Medical College, and Surgeon to the Philadelphia Hospital.

1878. Hess, Robert J., M.D.

1853. Hewson, Addinell, M.D.

1872. Hinkle, A. G. B., M.D.

1852. Hooper, William H., M.D.

1879. Hopkins, William Barton, M.D., Surgeon to the Out Department of the Pennsylvania, Episcopal, and University Hospitals, and Assistant Demonstrator of Surgery in the University of Pennsylvania.

1867. Horn, George H., M.D.

1868. Howell, Samuel B., M.D., Professor of Mineralogy and Geology in the University of Pennsylvania.

1882. Hughes, Daniel E., M.D.

1881. Huidekoper, Rush Shippen, M.D.

1854. Hunt, William, M.D., Surgeon to the Pennsylvania Hospital and to the Orthopædic Hospital.

1871. Hunter, Charles T., M.D., Demonstrator of Anatomy in the University of Pennsylvania, Surgeon to the Episcopal Hospital and Surgeon for Out-patients to the Pennsylvania Hospital.

1863. Hutchinson, James H., M.D., Physician to the Pennsylvania Hospital and to the Children's Hospital.

1871. Ingham, James V., M.D.

ELECTED

1864. Jones, S. P., M.D., Assistant Physician to the Pennsylvania Hospital for the Insane.

1867. Judson, Oliver A., M.D.

1877. Keating, John M., M.D., Obstetrician to the Philadelphia Hospital, Physician to St. Joseph's Hospital.

1849. Keating, William V., M.D., Physician to St. Joseph's Hospital.

1867. Keen, William W., M.D., Professor of Surgery in Woman's Medical College of Pennsylvania, Surgeon to St. Mary's Hospital, Consulting Surgeon to the Mission Hospital, and to the Philadelphia Home for Incurables.

*1844. King, Charles R., M.D.

1875. Kirkbride, J. J., M.D., Physician for Out-patients to the Pennsylvania Hospital.

1839. Kirkbride, Thomas S., M.D., LL.D., Physician-in-Chief to the Pennsylvania Hospital for the Insane.

1848. Klapp, Joseph, M.D., Physician to the Howard Hospital.

*1865. LaRoche, C. Percy, M.D.

1864. Leedom, John M., M.D.

1851. Leidy, Joseph, M.D., LL.D., Professor of Anatomy in the University of Pennsylvania.

1855. Lewis, Francis W., M.D.

1877. Lewis, Morris J., M.D., Physician to the Episcopal Hospital, Assistant Physician to the Children's Hospital, and to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1849. Lewis, Samuel, M.D.

1836. Littell, S., M.D., Consulting Surgeon to the Philadelphia Dispensary, Emeritus Surgeon to Wills Hospital.

1877. Longstreth, Morris, M.D., Lecturer on Pathological Anatomy in the Jefferson Medical College, Physician, and Pathologist and Curator, to the Pennsylvania Hospital.

1849. Ludlow, John L., M.D., Physician to the Philadelphia Hospital and to the Presbyterian Hospital.

ELECTED

1875. McCLELLAN, GEORGE, M.D., Surgeon to the Philadelphia Hospital.

1871. McFERRAN, J. A., M.D., Physician to the Gynæcological Hospital and Infirmary for Diseases of Children.

*1850. MAYER, EDWARD H., M.D.

1868. MEARS, J. EWING, M.D., Demonstrator of Surgery in the Jefferson Medical College and Gynæcologist to the Hospital of the same, Surgeon to St. Mary's Hospital.

1875. MEIGS, ARTHUR V., M.D., Physician to the Pennsylvania Hospital, and Assistant Physician to the Children's Hospital.

1881. MILLS, CHARLES K., M.D., Professor of Diseases of the Mind and Nervous System, in the Philadelphia Polyclinic and College for Graduates in Medicine, Lecturer on Mental Diseases and Electro-Therapeutics in the University of Pennsylvania, Neurologist to the Philadelphia Hospital.

1856. MITCHELL, S. WEIR, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases, Consulting Physician to the State Hospital for Women and Infants.

1882. MONTGOMERY, EDWARD E., M.D.

1863. MOREHOUSE, GEORGE R., M.D., Physician to St. Joseph's Hospital.

1839. MORRIS, CASPAR, M.D.

1883. MORRIS, HENRY, M.D.

1856. MORRIS, J. CHESTON, M.D., Consulting Physician to the Mission Hospital.

1861. MORTON, THOMAS G., M.D., Surgeon to the Pennsylvania Hospital and to the Orthopædic Hospital, Consulting Surgeon to the Jewish Hospital, Emeritus Surgeon to Wills Hospital.

1864. MOSS, WILLIAM, M.D.

1882. MUSSER, JOHN H., M.D., Chief of the Medical Dispensary of the Hospital of the University of Pennsylvania.

1865. NEBINGER, ANDREW, M.D., Physician to St. Mary's Hospital.

ELECTED

1869. Norris, Herbert, M.D., Physician to the Catharine Street Dispensary.

1865. Norris, Isaac, Jr., M.D.

1870. Norris, John C., M.D.

1866. Norris, William F., M.D., Clinical Professor of Diseases of the Eye in the Hospital of the University of Pennsylvania, Surgeon to Wills Hospital.

1858. Packard, John H., M.D., Surgeon to the Episcopal Hospital and to St. Joseph's Hospital.

1864. Pancoast, William H., M.D., Professor of General, Descriptive, and Surgical Anatomy in the Jefferson Medical College, Surgeon to the Philadelphia Hospital, Consulting Surgeon to the Charity Hospital and to the Pennsylvania Free Dispensary for Skin Diseases.

1882. Parish, William H., M.D., Professor of Anatomy in the Woman's Medical College, and Obstetrician to the Philadelphia Hospital.

*1854. Parrish, Joseph, M.D.

1854. Penrose, R. A. F., M.D., LL.D., Professor of Obstetrics and Diseases of Women and Children in the University of Pennsylvania, Consulting Obstetrician to the State Hospital for Women and Infants, Visiting Physician to the Preston Retreat.

1868. Pepper, William, M.D., LL.D., Professor of Clinical Medicine in the University of Pennsylvania, Physician to the Philadelphia Hospital and to the Children's Hospital, Consulting Physician to the Mission Hospital.

1883. Piersol, George A., M.D., Assistant Demonstrator of Normal Histology in the University of Pennsylvania.

1872. Porter, William G., M.D., Surgeon to the Presbyterian Hospital and to the Philadelphia Hospital.

1883. Randolph, N. Archer, M.D., Assistant Demonstrator of Physiology in the University of Pennsylvania.

1866. Reed, Thomas B., M.D., Surgeon to the Presbyterian Hospital, Microscopist to the Philadelphia Hospital.

ELECTED

1842. Reese, John J., M.D., Professor of Medical Jurisprudence in the University of Pennsylvania, Physician to St. Joseph's Hospital, and to the Gynæcological Hospital and Infirmary for Diseases of Children.

1871. Richardson, Elliot, M.D., Lecturer on Practical Obstetrics in the University of Pennsylvania, Surgeon for Out-patients to the Pennsylvania Hospital, Obstetric Physician to the Philadelphia Dispensary, Physician to the Catharine Street Dispensary.

1869. Richardson, Joseph G., M.D., Professor of Hygiene and Demonstrator of Histology in the University of Pennsylvania, Physician to the Presbyterian Hospital, Physician for Out-patients and Microscopist to the Pennsylvania Hospital.

*1857. Richardson, Tobias G., M.D.

1882. Roberts, A. Sidney, M.D.

1878. Roberts, John B., M.D.

1843. Rodman, Lewis, M.D., Visiting Physician to the Preston Retreat.

1857. Rogers, Robert E., M.D., LL.D., Professor of Chemistry in the Jefferson Medical College.

1838. Ruschenberger, W. S. W., M.D., Medical Director U. S. N., retired.

*1852. Sargent, Fitz William, M.D.

*1864. Sargent, Winthrop, M.D.

1866. Schaffer, Charles, M.D.

1870. Schell, Henry S., M.D., Surgeon to Wills Hospital, Ophthalmic and Aural Surgeon to the Children's Hospital.

1875. Seyfert, Theodore H., M.D., Physician to the Gynæcological Hospital and Infirmary for Diseases of Children.

1877. Shakespeare, Edward O., M.D., Lecturer on Refraction and Accommodation of the Eye, and on Operative Ophthalmic Surgery, in the University of Pennsylvania, Pathologist and Ophthalmologist to the Philadelphia Hospital.

1868. Shapleigh, E. B., M.D.

ELECTED

1882. Sheppard, Frederick C., M.D., Assistant Gynæcologist to the Hospital of the University of Pennsylvania, and Surgeon for Out-patients to the Children's Hospital.

1876. Shippen, Edward, M.D., U. S. N.

1880. Simes, J. H. C., M.D., Professor of Genito-Urinary and Venereal Diseases in the Philadelphia Polyclinic and College for Graduates in Medicine, and Assistant Surgeon to the Episcopal Hospital.

1873. Simpson, James, M.D., Physician to St. Mary's Hospital.

1872. Sinkler, Wharton, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1863. Smith, Albert H., M.D., Physician to and Lecturer on Obstetrics in the Philadelphia Lying-in Charity, Visiting Physician to the Preston Retreat, Consulting Physician to the Hospital of the Good Shepherd, Radnor.

1863. Smith, A. K., M.D., U. S. A.

1864. Smith, Edward A., M.D.

*1856. Smith, R. K., M.D.

1864. Spooner, Edward A., M.D.

1875. Starr, Louis, M.D., Lecturer on General Symptomatology in the University of Pennsylvania, Physician to the Episcopal Hospital and Assistant Physician to the Children's Hospital.

1842. Stillé, Alfred, M.D., LL.D., Professor of the Theory and Practice of Medicine in the University of Pennsylvania, Consulting Physician to the State Hospital for Women and Infants.

1846. Stocker, Anthony E., M.D.

1871. Strawbridge, George, M.D., Clinical Professor of Diseases of the Ear in the Hospital of the University of Pennsylvania, Surgeon to Wills Hospital, Ophthalmic Surgeon to the Presbyterian Hospital, Surgeon to the Eye and Ear Department of the Philadelphia Dispensary.

1855. Stroud, William D., M.D.

1867. Taylor, R. R., M.D.

ELECTED

1867. Thomas, Charles H., M.D., Surgeon to the Philadelphia Hospital.

1869. Thomson, William, M.D., Honorary Professor of Ophthalmology in the Jefferson Medical College, and Ophthalmic Surgeon to the Hospital of the same.

*1854 Tilden, W. P., M.D.

1870. Turner, A. Paul, M.D.

1866. Tyson, James, M.D., Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania, Physician to the Philadelphia Hospital.

*1852. Tyson, James L., M.D.

1864. Vandyke, E. B., M.D.

1873. Van Harlingen, Arthur, M.D., Consulting Physician to the Dispensary for Skin Diseases.

1883. Vinton, Charles Harrod, M.D.

1852. Wallace, Ellerslie, M.D., Emeritus Professor of Obstetrics and Diseases of Women and Children in the Jefferson Medical College, Consulting Ob-tetrician to the State Hospital for Women and Infants, Visiting Physician to the Preston Retreat, Consulting Physician to the Lying-in Department of the Northern Dispensary.

1873. Wallace, W. H., M.D.

*1839. Warrington, Joseph, M.D.

Webb, William H., M.D.

Welch, William M., M.D., Physician to Municipal Hospital for Contagious Diseases.

1878. White, J. William, M.D., Demonstrator of Surgery and Lecturer on Venereal Diseases in the University of Pennsylvania, Surgeon to the Philadelphia Hospital.

1880. Willard, DeForest, M.D., Lecturer on Orthopædic Surgery in the University of Pennsylvania, Surgeon to the Presbyterian Hospital.

*1878. Williamson, Jesse, M.D.

ELECTED.

1851. Wilson, Ellwood, M.D., Consulting Physician to the Philadelphia Lying-in Charity, Visiting Physician to the Preston Retreat.

1881. Wilson, H. Augustus, M.D.

1874. Wilson, J. C., M.D., Lecturer on Physical Diagnosis in the Jefferson Medical College and Physician to the Hospital of the same, Physician to the Philadelphia Hospital.

1871. Wistar, Thomas, M.D.

1848. Wister, Caspar, M.D., Consulting Physician to the Philadelphia Dispensary.

1852. Wister, Owen Jones, M.D., Consulting Surgeon to the Germantown Hospital.

1865. Wood, Horatio C., M.D., Professor of Materia Medica, Pharmacy and General Therapeutics in the University of Pennsylvania and Clinical Professor of Diseases of the Nervous System in the Hospital of the same.

1880. Woodbury, Frank, M.D., Physician to the German Hospital.

1866. Woods, D. F., M.D., Physician to the Presbyterian Hospital.

1878. Wormley, Theodore G., M.D., LL.D., Professor of Chemistry in the University of Pennsylvania.

1860. Wurts, Charles Stewart, M.D.

1861. Yarrow, Thomas J., M.D.

*1840. Zantzinger, William S., M.D.

[It is particularly requested that any change of appointment, etc., may be communicated to the Committee of Publication before the first of July, in each year, in order that the List may be made as correct as possible.]

CORRESPONDING FELLOWS.

ELECTED

1880. Carrow, Flemming, M.D., Canton, China.

1880. Chiara, Domenico, Milan, Italy.

1880. Wasseige, Adolphe, Liège, Belgium.

ASSOCIATE FELLOWS.

[Limited to Fifty, of whom Twenty may be Foreigners.]

ELECTED

1873. Acland, Henry W., M.D., F.R.S., Oxford, England.
1869. Alvarenga, P. F. Da Costa, Lisbon, Portugal.
1847. Atlee, John L., M.D., LL.D., Lancaster, Pennsylvania.
1876. Baldwin, W. O., M.D., Montgomery, Alabama.
1876. Barker, Fordyce, M.D., New York.
1877. Barnes, Robert, M.D., London, England.
1876. Bigelow, Henry J., M.D., Boston, Massachusetts.
1876. Billings, John S., M.D., U. S. A., Washington, District of Columbia.
1876. Bowditch, Henry I., M.D., Boston, Massachusetts.
1865. Butcher, R. G. H., M.D., Dublin, Ireland.
1877. Byford, William H., M.D., Chicago, Illinois.
1877. Chaillé, Stanford E., M.D., New Orleans, Louisiana.
1876. Clark, Alonzo, M.D., New York.
1876. Comegys, C. G., M.D., Cincinnati, Ohio.
1876. Corson, Hiram, M.D., Norristown, Pennsylvania.
1881. Courty, Amédée, Montpellier, France.
1876. Davis, N. S., M.D., Chicago, Illinois.
1876. Donaldson, F., M.D., Baltimore, Maryland.
1868. Flint, Austin, M.D., LL.D., New York.
1878. Fothergill, J. Milner, M.D., London, England.
1876. Green, Traill, M.D., Easton, Pennsylvania.
1868. Hamilton, Frank H., M.D., LL.D., New York.

ELECTED

1874. Jackson, J. Hughlings, M.D., London, England.
1876. Johnson, George, M.D., F.R.S., London, England.
1876. Johnston, Christopher, M.D., Baltimore, Maryland.
1876. Jones, Joseph, M.D., New Orleans, Louisiana.
1876. King, James, M.D., Pittsburgh, Pennsylvania.
1876. Kinloch, R. A., M.D., Charleston, South Carolina.
1877. Lister, Joseph, LL.D., F.R.S., London, England.
1865. Macleod, G. H. B., M.D., Glasgow, Scotland.
1876. Moore, E. M., M.D., Rochester, New York.
1876. Mowry, R. B., M.D., Allegheny City, Pennsylvania.
1873. Ogle, John W., M.D., London, England.
1874. Paget, Sir James, Bart., D.C.L., LL.D., F.R.S., London, England.
1842. Parker, Peter, M.D., Washington, District of Columbia.
1876. Parker, Willard, M.D., New York.
1876. Pollock, A. M., M.D., Pittsburgh, Pennsylvania.
1876. Porcher, F. Peyre, M.D., Charleston, South Carolina.
1869. Valcourt, Th. de, Cannes, France.
1857. Valery, Gaetano, Florence, Italy.
1861. Velasco, Pedro Gonzales, Madrid, Spain.
1860. Walther, Hermann, Dresden, Saxony.

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NOTICE.

The present volume of Transactions contains the papers read before the College from December, 1881, to July, 1883, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its Transactions.

[Volume VI. of the Third Series of the Transactions has been edited by the Recorder, Dr. J. Ewing Mears.]

MEMOIR

OF

H. LENOX HODGE, A.M., M.D.,

LATE DEMONSTRATOR OF ANATOMY IN THE UNIVERSITY OF PENNSYLVANIA, SURGEON TO THE CHILDREN'S AND TO THE PRESBYTERIAN HOSPITALS.

By

WILLIAM G. PORTER, M.D.

[Read October 4, 1882.]

Hugh Lenox Hodge was born in Philadelphia July 30th, 1836, his parents being the late Professor Hugh L. Hodge and Margaret Elizabeth Aspinwall Hodge. His family resident in this country since 1730, being of Scotch-Irish, English-Welsh, and Huguenot descent.

Andrew Hodge, the founder of the family in this country, came to Philadelphia from the north of Ireland, and established himself in business as a merchant. Three sons were born to him, Andrew, James, and Hugh. The first became a merchant; the second, a sea captain; and the third, Hugh, studied medicine, and, after receiving his diploma, served as a surgeon in the Continental Army during the War of Independence. At the termination of the war he returned to Philadelphia, and established himself in practice. In 1790 he married Maria Blanchard, a Boston lady of Huguenot descent. In the dreadful epidemics of yellow fever which visited Philadelphia in 1793 and 1795 Dr. Hugh Hodge bore

a distinguished part. He died in 1798, leaving two sons, Hugh L. and Charles Hodge—the one afterwards so distinguished as Professor of Obstetrics in the University of Pennsylvania, the other as Professor in the Theological Seminary at Princeton. Lenox Hodge, it will thus be seen, naturally came by a taste for the medical profession, his father and grandfather before him having been distinguished medical men.

He was educated at private schools, and entered the Academical Department of the University of Pennsylvania in 1851, graduating in 1855 the first in his class, and delivering the Greek salutatory oration at the commencement. He at once began the study of medicine with his father, and received the degrees of A.M. and M.D. in 1858. Soon after this he was elected one of the Resident Physicians to the Pennsylvania Hospital, and served his full term in that institution.

He began private practice in 1860, at the old family mansion N. W. corner of Ninth and Walnut streets. In 1861 he was appointed Demonstrator of Surgery and Chief of the Surgical Clinic and Dispensary in the University of Penna. In the same year, in connection with Drs. Bolling and J. Cheston Morris, he started a Quiz Association which afterwards developed into the Medical Institute. In 1879 he was appointed Demonstrator of Anatomy in the University, having resigned his position as Demonstrator of Surgery several years before, and having opened a private school of Anatomy and Surgery on Chant Street, which he relinquished on receiving his appointment as Demonstrator of Anatomy.

During the war he was one of the surgeons to the U. S. Satterlee Hospital in West Philadelphia, and was also attached to the Pennsylvania Reserve Corps of Surgeons, and

rendered valuable aid in the field at Yorktown, White House, Chambersburg, Fredericksburg, and Gettysburg.

He was appointed Surgeon to the Children's Hospital in 1864, and to the Presbyterian Hospital on its organization in 1872. He was a member of the American Medical Association, of the State Medical Society of Pennsylvania, of the Philadelphia County Medical Society, of the Obstetrical and Pathological Societies, being President of the latter for three years, and a Fellow of this College. He was one of the Councillors of the College for several years before his death. He was also Consulting Surgeon to several charitable institutions in this city.

When the Society for the Organization of Charity and the Suppression of Mendicancy was organized he felt a great interest in it, and took an active part in its work. He was one of the Directors of the Seventh Ward branch of the Association, and President of its Assembly. Of a deeply religious turn of mind he had been a member of the Second Presbyterian Church and one of its Elders for many years, and when the old church building on Seventh Street was sold, and the congregation decided to remove to the new building site at Twenty-first and Walnut streets, he was one of the Building Committee, and a liberal contributor and active worker in securing the funds necessary.

He was married January 7th, 1869, to Harriet Roosevelt, daughter of Charles W. Woolsey, of New York.

Having thus briefly outlined the life-work of our friend, let us now examine it a little more in detail.

Lenox Hodge was born with great medical opportunities. The third of his race and name to practice medicine in Philadelphia, his father a Professor in the Medical Department of the University, he had many opportunities which

were denied to others less fortunately situated. Many men under these circumstances would have neglected or failed to appreciate their advantages. Not so with Hodge; he was ambitious and conscientious, and he devoted himself to his profession with a singleness of purpose that soon bore fruit.

Early in his professional career he decided to make a specialty of Surgery and the Surgical Diseases of Women, and in 1861 began to teach his profession. In conjunction with Drs. Bolling and J. Cheston Morris, in the spring of that year, he started a Quiz Association, whose object was to examine the students of the University of Pennsylvania on the lectures delivered by the professors in the medical department of that institution during the winter months. This Association was afterwards known as the Medical Institute, a title first given in this city, so far as can be ascertained, by Dr. Chapman to a class organized by him, and borne, with a few intervals, by various organizations from that time. In 1863 Dr. Morris resigned his position, and Dr. James H. Hutchinson was appointed in his place. The Medical Institute began in 1861 with four students, and from that time until 1872, when it disbanded, four hundred and ninety-eight students were enrolled on its books: the largest class in any one year, that of '68 and '69, contained eighty-five students. Dr. Bolling examined the students in Materia Medica; Dr. Hutchinson in the Practice of Medicine and Chemistry; and Dr. Hodge in Anatomy, Physiology, Surgery, and Obstetrics. In addition to the daily examinations on the University Lectures, Special Lectures were given by Drs. Hodge and Hutchinson once a week on subjects not lectured on during the term by the professors in the University. Hodge, it will thus be seen, examined during the winter months on four branches. As an instructor he was remarkable alike for his

promptness, patience, and power of imparting knowledge. In three years, while a pupil of the Institute, I never knew him to be absent at the appointed time from his post. Meeting the students so frequently as he did it was perfectly natural that he should be the favorite, and that the hours on which they met him should have been anticipated with peculiar pleasure.

During the months of April, May, June, and September, daily examinations were held on the various text-books, and lectures were given, and hospital instruction furnished by Dr. Hodge at the Children's, and Dr. Hutchinson at the Episcopal Hospitals. Hodge lectured and quizzed on surgery; lecturing for three-quarters of an hour, and reviewing the previous lecture by a quiz of fifteen minutes. His lectures were clear, practical, and instructive, and always showed great care in preparation, were abundantly illustrated, and the subjects were generally chosen because they were seldom, if ever, alluded to in the surgical lectures at the University. In addition to the examinations and lectures on surgery, he always examined the class in obstetrics, the text-book being Hodge's Obstetrics, in the preparation of which he had so large a share. The preface to the work concludes as follows: "The superintendence of the illustrations and the laborious duties of editor have chiefly devolved on the author's son, H. Lenox Hodge, M.D., without whose assistance this work would probably never have been completed."

After a prosperous career of eleven years the Medical Institute was disbanded in 1872, and from that time until his death he was preceptor for forty office students.

In the welfare of the Presbyterian Hospital he took a most active interest. He was a member of each of the

Building Committees which had charge of the construction of the Surgical Pavilion Wards. And in addition to liberal contributions of money he gave most freely of his time and experience in all the details of planning and building. In addition to his regular term of service in the surgical wards of three months in each year, he almost always had private patients of his own in the wards and in the private rooms of the hospital; in fact his duties of one kind or another called him to the hospital almost daily throughout the year. As a surgeon he was perfectly fearless and perfectly conscientious in everything. He never came to conclusions hurriedly. He gave to each case that calm, deliberate, enlightened, and unbiased judgment to which it was entitled. I have seen him in as trying emergencies as it is possible for a surgeon to be placed, and I never knew him to falter for an instant. A neat operator and careful dresser; like all true surgeons he paid that great attention to little things which can alone secure success. He possessed in a remarkable degree the power of inspiring the confidence and affection of his patients. His kindly manner, his devotion alike to the humblest as well as the most affluent of his patients, won for him the lasting friendship of all. How often have I heard from his former patients, the sentiment we have lost not only a physician, but a friend. He never seemed to be in a hurry with them, and no matter how much he was pressed for time he always had an attentive ear for all their suggestions or complaints. His very presence in the sick-room made the invalid more contented, and I can speak from personal experience of the pleasure and comfort his visits in sickness were.

He had received from his father a number of patients, and his church connections had brought him others who had

been attended by him for years without fee, simply because they were too poor to pay and were known to be deserving. Many of them were persons who had not always been in such straitened circumstances, and had been made dependent by various misfortunes; but he always manifested the same interest in them that he did in his very best patients, and no care or trouble seemed to be too great for him to assume if, by any means, he could add to their comfort or happiness. Few had any idea of what constant calls of this kind were made on his time and attention, and how cheerfully and sympathetically they were always answered. His connection with the Second Presbyterian Church has been already alluded to; an elder of his church, a member of the Building Committee of the new church; his religious duties were as exacting almost as his medical. Religion with him was not a mere cloak to be put on or off at pleasure, it was an essential part of his life. He attended the services of his church whenever possible, and he almost always made it possible, and was in every position of trust or duty a thorough and systematic worker. His views, the result of early training, of honest belief and positive conviction, were followed through life consistently and faithfully.

When in 1870 he was appointed Demonstrator of Anatomy in the University he transformed the old anatomical room into a thoroughly lighted, well-ventilated, and handsomely furnished dissecting-room. A daily record of all anomalies found in the room was kept, and an annual prize was offered by him to stimulate the students in their anatomical studies. His views were embodied in a little pamphlet, entitled Anatomical Rooms, a plea for their construction, ventilation, and hygienic management, which was the result of his experience in Chant Street and at the University, and the exten-

sive examination of the principal anatomical rooms both of this country and of Europe.

Dr. Hodge was a frequent attendant on the various medical societies of which he was a member, and a frequent contributor to the literature of his profession. His share in the production of Hodge's Obstetrics has been already alluded to. The article on Compression of the Fœtal Head by the Forceps and Cephalotribe, by Professor Hugh L. Hodge, was completed by him. He delivered the address on Surgery before the State Medical Society at its annual session held in Harrisburg, in June, 1877, besides writing numerous papers on Excision of the Hip, Excision of the Knee, Subcutaneous Osteotomy, Kolpo-Cystotomy, Metallic Suture, Tracheotomy for Pseudo-membranous Croup, and the Drainage of Abscesses and Wounds by Solid Metallic Probes; he also prepared a Note-book for Cases of Ovarian Tumors and other Abdominal Enlargements, a pamphlet on Anatomical Rooms, suggested a modification of Physick's long splint for the treatment of fracture of the femur, and invented a Canula for tapping Ovarian Cysts. The nature of his specialty and his long connection with medical teaching had entailed a burdensome correspondence, which was often kept within bounds only by depriving himself of hours of much-needed repose after the toilsome duties of the day.

Dr. Hodge was an unsuccessful candidate for the position of Visiting Surgeon to the Philadelphia Hospital and of Professor of Clinical Surgery in the University of Pennsylvania, made vacant by the death of Professor John Neill. In March, 1875, he removed to his new house, No. 506 South Broad Street. It was a model of arrangement, of comfort, and of elegance. He had superintended all the details of its construction himself, and everything betokened a long and

prosperous career. Happy in his domestic relations with a loving wife, a son to bear his name, devoted to his profession with a rapidly increasing practice, and the respect of his fellow-men, everything looked bright and promising, but on May 11, 1878, but little more than three years after his removal, his wife suddenly died. He had left her in the morning slightly indisposed, she was near her confinement, and had complained of heart-symptoms for some time, but there was nothing apparently alarming in her condition. He returned in the midst of his morning rounds a short time afterwards to find her a corpse. From the terrible shock of his wife's death Hodge recovered but slowly. Completely crushed at first he soon sought forgetfulness of his sorrow in work, and under the calm, dignified exterior of the man few suspected how profoundly he had been shaken. Work which had been a pleasure to him before became a necessity now, and he really seemed to have no leisure, and to give himself no rest. Most physicians, in active practice, find their time thoroughly occupied by their professional calls, and the necessary amount of reading and study, but when, in addition to the cares of a practice, we have added his duties as a teacher, as a surgeon to two hospitals, a large correspondence, and his religious and extra-professional duties, it will readily be understood that he was habitually overworking himself. Endowed by nature with a strong constitution, he withstood an amount of toil which would have broken down a less vigorous man—with a laborious practice, with constant, unceasing, ever-pressing calls on his time and strength—he knew no rest. Overworked for years, at last the inevitable result followed. He did not seem at first to understand that he was ill. He had never been sick since childhood he used to say. I received a note from him one

stormy morning, asking me to see some patients for him, and saying that he was indisposed. I called at once upon him, and found him at work with some papers in his office. He was haggard and worn, and so oppressed for breath that it was painful to talk with him. The night before, though suffering from a severe cold, he had presided at a meeting of the Charity Organization Society, and on his way home was so oppressed that he had to stop at a hotel to rest. I urged him to see a physician, but he made light of it, although admitting that for some time he had been troubled with palpitation of the heart at night. This symptom appeared soon after the shock he experienced on the death of his wife, and he then told me that he had never from that time been physically the man he was before. As he expressed it, "I can go upstairs two steps at a time without getting out of breath, but I have had ever since that time what I never had before, palpitation of the heart and oppression at night." Days went by without improvement. The servants told me that he had no rest at night, and finally, from sheer exhaustion, he had to remain in his room. Dr. John F. Meigs was called to see him, and found a laboring, over-acting heart, with evidence of mitral disease and congestion of the lungs. Confined at first to the lounge, and then to his bed, a long, tedious, and suffering illness awaited him. Surrounded by his family, and constantly attended by his physicians and friends, he struggled manfully against his disease for weeks. Throughout his long illness, although suffering almost constantly, he was patient, courageous, and always considerate of, and solicitous for, the comfort of those who were waiting on him. So much so was this the case that he would never allow his medical friends, many of whom gladly volunteered, to remain with him at night, and, although for weeks some

physician was always in the house at night in case of an emergency, this fact had to be withheld from him. For days and weeks he struggled on, but his symptoms gradually, but surely, became worse, and on June 16, 1881, he died.

"His life was gentle; and the elements
So mixt in him that nature might stand up,
And say to all the world—This is a man!"

REMARKS MADE BY THE PRESIDENT,

ALFRED STILLÉ, M.D., LL.D.,

On assuming the Chair, February 7, 1883.

GENTLEMEN—FELLOWS:—

During my forty years of Fellowship in the College I have had the honor of serving it in nearly all of its offices and committees. To my great contentment, as well as my pride, it has seen fit to complete my crown of honor by choosing me to preside over its deliberations. I am most grateful for this distinction, which affords me more satisfaction than any other I have ever received, because it is conferred by those who for many years have been in a position to judge of my conduct and motives. But in proportion to the honor of the office is my dread of not discharging its duties correctly and acceptably. My notion of a presiding officer is that while he is, first of all, the mouth-piece of the body that elected him, and while his conduct should be distinctly traced over the letter of its laws, he should, at the same time, be animated by a desire to quicken the zeal and activity of its members in attaining the objects for which it was created.

He must, in that case, constantly look to his fellows for counsel and support, not only in the performance of his routine duties, but in carrying out measures that may be undertaken to promote their professional culture, and the influence in the community of the institution over which he presides.

No other agencies can do so much towards enlightening the scientific ignorance and correcting the false ethical standards that unfortunately prevail in the medical profession. None other can so well enlighten the laity in regard to the value of the highest possible grade of professional culture, and in regard also to the countless blessings that a thoroughly educated medical profession confers upon the public.

The day is passing when it is any longer necessary to hold a dark screen between physicians and educated laymen, and the

better the world shall learn what are the aims and the achievements of a physician's life, the sooner will be dispelled the prejudices that antagonize the medical profession and the delusions under which the public become the victims of error and of fraud.

Methods have already been devised, which will be duly laid before you, and generous assistance has been pledged, for carrying out these purposes. There can be no doubt that they will afford an opportunity, such as never before arose, to widen and strengthen the influence of the College. But neither these nor any material agents can accomplish for it the greatest possible good, unless all of its Fellows are aroused by the thought of the great future that is dawning upon it; and attest, by a more active participation in its labors, their devotion to the purposes for which it was created.

Permit me, then, Fellows of the College, not only to invite but to urge your mutual coöperation in the work that lies before us. This is the fitting place, and it is the only one, where physicians chosen out of the whole number by reason of their exceptional merit can meet together upon neutral ground, and, laying aside all selfish and narrow motives, devote themselves to those objects which the charter of the College sets forth, the first of which is "to advance the science of medicine and thereby lessen human misery;" and the last of which is to "cultivate order and uniformity in the practice of physic."

REPORT

OF THE

COMMITTEE ON METEOROLOGY AND EPIDEMICS FOR THE YEAR 1880.

By

RICHARD A. CLEEMANN, M.D.,

MEMBER OF THE BOARD OF HEALTH OF PHILADELPHIA.

[Read December 7, 1881.]

Meteorology.

Temperature.—The mean annual temperature of the year 1880, as calculated from the monthly means, was 54.6° F. (Table A), or 1.7° F. above the average for the nine years 1872–1880: the year was, then, taken as a whole, a warm one; indeed, during these nine years, there was but one for which a higher mean temperature was recorded, 1878, when 54.7° F. was attained (Table B).

The first six months of the year and September each exceeded their respective averages of mean temperature during nine years. January, with the mean reading of the thermometer for the month at 41° F., went beyond its average as much as 8.7° F., and the mean of 39.1° F. for February was 6.1° F. above its average; so that the winter was wonderfully mild for this climate. A greater warmth of the spring was not so marked, except at the close, the monthly means

TABLE A.—METEOROLOGICAL ELEMENTS OF THE CLIMATE AT PHILADELPHIA FOR 1880.

Furnished by E. F. Townsend, Observer, Signal Service, U. S. A.

Date, 1880.	BAROMETER. Mean of Telegraphic observations. Local observations.	Corrected for temp., instrumental error, and elevation. A. M.	P. M.	Mid-night.	Corrected for temperature and instrumental error only. A. M.	P. M.	Mid-night.	Range. Highest.	Lowest.	Difference.	THERMOMETER. Mean of Telegraphic observations. Local observations.	A. M.	P. M.	Mean of daily extremes.	Range. Maximum	Minimum	Difference	Mean relative humidity, based on 8 A.M. and 8 P.M. observations.	WIND. Prevailing direction.	Number of Miles. N. or S. to 8 P.M.	8 P.M. to midnight.	Midnight to 8 A.M.	8 A.M. to noon.	Total.	Max. mean velocity during the month.	Amount of rain or melted snow. inches and hundredths.	No. of days on which rain or snow fell.	Number of auroras.
Jan.	30.190	30.213	30.167	30.195	30.159	30.114	30.142	30.744	29.559	1.155	41.0	36.8	46.5	40.5	63	19	44	75.0	N. E.	1842	1531	1612	1743	6728	32	1.54	12	0
Feb.	30.120	30.122	30.089	30.144	30.068	30.036	30.091	30.637	29.163	1.474	39.1	34.9	44.9	37.0	67	12	55	68.4	N. W.	2227	1671	1415	2443	8256	48	2.43	10	0
March	30.067	30.090	30.025	30.080	30.037	29.973	30.027	30.498	29.348	1.150	39.6	35.5	44.2	38.5	75	20	25	67.3	N. E.	2825	2120	2092	2606	9643	40	3.73	14	0
April	30.022	30.059	29.987	30.020	30.008	29.937	29.969	30.374	29.543	0.831	52.4	48.2	58.8	50.2	81	29	52	57.1	S. W.	3083	2208	1700	2633	9624	41	2.43	6	0
May	30.063	30.101	30.027	30.065	30.051	29.978	30.012	30.362	29.764	0.598	68.8	62.0	77.7	65.3	96	36	60	59.0	S. W.	2104	1600	1883	2720	8307	34	0.54	4	0
June	29.999	30.026	29.969	30.004	29.977	29.921	29.955	30.291	29.652	0.639	73.8	68.7	80.2	70.4	94	51	43	62.6	W.	2144	1637	1289	1778	6848	32	1.87	2	0
July	29.993	30.016	29.969	29.994	29.967	29.921	29.945	30.196	29.688	0.508	75.6	70.2	81.7	72.5	95	60	35	65.2	S. W.	1860	1632	1417	1413	6322	27	7.74	8	0
Aug.	30.058	30.090	30.045	30.081	30.041	29.993	30.032	30.447	29.729	0.718	72.8	75.3	79.5	70.7	90	56	34	70.2	S. W.	1882	1526	1240	1691	6339	25	5.00	7	2
Sept.	30.061	30.086	30.022	30.071	30.036	29.973	30.021	30.379	29.650	0.729	67.7	62.9	75.1	65.1	91	50	41	67.0	N. W.	1908	1351	1382	1745	6386	30	1.10	3	0
Oct.	30.123	30.155	30.087	30.121	30.103	30.037	30.070	30.448	29.484	0.964	55.1	50.0	61.7	52.5	82	35	47	67.5	S. W.	2153	1453	1329	1938	6870	36	1.74	12	0
Nov.	30.253	30.275	30.224	30.253	30.227	30.172	30.200	30.775	29.546	1.229	40.5	35.9	45.1	39.7	69	10	59	67.9	N. W.	2258	1652	1614	1769	7293	32	1.75	13	0
Dec.	30.048	30.092	30.012	30.068	30.007	29.959	30.014	30.508	29.655	0.853	28.6	26.0	32.1	27.5	52	—5	57	74.3	N. W.	2685	2357	2254	2239	9535	36	4.07	11	0
Sums	361.000	361.295	360.023	361.096	360.681	360.014	360.178	365.629	354.781	10.848	655.0	600.4	726.5	624.7	940	37	547	80.15										
Ann. means	30.092	30.108	30.052	30.091	30.057	30.001	30.040	30.469	29.565	0.904	54.6	50.5	60.5	52.5	79	6.1	1 45.6	66.8						92211		33.48	102	2

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of temperature in the several months being for March 39.6° F., for April 52.4° F., and for May 68.8° F., or respectively .7° F., 3.3° F., and 6.9° F. above their averages. So high a degree of heat in May was very exceptional, the mean temperature of the month in no one of the previous eight years rising within 7° F. of this value. On the other hand, the months in the latter half of the year, with the exception of September, already mentioned, were each of lower temperature than usual, December being very cold. The fact that June, marking a mean of 73.8° F., exceeded its average 2.3° F., while July, recording 75.6° F., and August 72.8° F., were respectively 1° F. and .6° F. below their means, led to the summer being more equable than we are accustomed to find it. The fall, on the contrary, showed a greater variation in temperature between the months than is common, the 67.9° F. of mean temperature of September being 1.6° F. above its average, while the 55.1° F. of October and the 40.5° F. of November were respectively .9° F. and 2° F. below their means. The mean temperature of December, 28.6° F., is as much as 5.4° F. below its average for nine years, and is, with the exception of that for 1876, the lowest recorded for the month during that period.

The range of the thermometer during the year was through 101° F. of temperature; from 96° F. recorded in May to –5° F. noted in December. The month of May was distinguished by the widest variation in temperature, the thermometric range being through 60° F., and August by the narrowest, 34° F.; between these two the other months came in the following order: July with a range of 35° F., September of 41°

TABLE B.—COMPARISON OF METEOROLOGICAL ELEMENTS OF THE CLIMATE AT PHILADELPHIA, FOR THE YEARS 1872, 1873, 1874, 1875, 1876, 1877, 1878, 1879, AND 1880, WITH MEANS.

	Meteorological elements	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.	Mean of 9 years.
March.											
January.	Thermometer. Mean.	29.4	28.9	36.7	26.1	37.7	28.4	32.7	29.9	41.0	32.3
	Thermometer. Range.	43.0	60.0	55.5	49.5	52.0	41.0	40.0	59.0	44.0	49.3
	Mean barometer.	30.053	30.113	30.193	30.231	30.161	30.063	30.075	30.069	30.190	30.128
	Mean humidity.	....	....	70.7	71.0	70.0	76.0	77.0	72.3	75.0	72.8
	Rainfall.	.95	5.84	4.53	2.83	1.52	2.62	3.94	2.73	1.51	2.93
February.	Thermometer. Mean.	31.2	29.8	33.2	26.1	34 0	36 9	36.6	29.9	39.1	33.0
	Thermometer. Range.	45.0	50.0	64.0	58.0	56.0	44.0	46.0	52.0	55.0	52.0
	Mean barometer.	30.011	30.016	30.174	30 131	30.131	30.084	29.996	30.087	30.120	30.081
	Mean humidity.	....	....	67.7	66.0	69 0	64.0	72.0	71.3	68.4	67.9
	Rainfall.	1.12	4.75	2.46	3.20	5.03	0.84	1.64	1.19	2.43	2.52
March.	Thermometer. Mean.	32.1	39.1	40.0	34 9	37.4	38.2	46.4	41.3	39.6	38.7
	Thermometer. Range.	52.0	53.0	48.0	49.0	57.0	52.0	53.0	50.0	55.0	52.2
	Mean barometer.	30.054	30.001	30.022	30.104	30.040	30.001	29.984	30.108	30.067	30.040
	Mean humidity.	....	....	64.0	69.0	68.0	68.0	66.0	69.6	67.3	67.2
	Rainfall.	3.67	2.04	2.16	3.10	6.71	3.40	2.89	2.28	3.53	3.31
April.	Thermometer. Mean.	51.6	45.3	42.5	45.5	49 1	50.0	55.9	49 1	52.4	49.1
	Thermometer. Range.	54.0	35.0	46.5	51.0	44.0	50.0	37.0	55.0	52.0	47.1
	Mean barometer.	30.063	29.947	30.055	29.978	30.004	29.983	29.809	29.935	30.022	29.977
	Mean humidity.	....	47.5	72.2	59.0	53.0	60 0	66.0	63.3	57.1	59.6
	Rainfall.	2.60	3.51	9.76	2.85	2.16	2.66	2.55	4.21	2.43	3.63
May.	Thermometer. Mean.	61.5	59.6	59.9	61.4	60.9	60.7	61.0	63.6	68.8	61.9
	Thermometer. Range.	43.0	45.0	51.0	49.0	47 0	49.0	41.0	49.0	60.0	48.0
	Mean barometer.	29.975	30.009	29.982	29.993	30.064	29.997	29.939	30.054	30.063	30.008
	Mean humidity.	....	60.3	57.7	54.0	62.0	54.0	62.0	64.5	59.0	59.0
	Rainfall.	2.15	5.83	2.75	1.36	4.45	1.10	3.29	1.22	0.54	2.52
June.	Thermometer. Mean.	72.7	70.6	73.0	70.5	74.2	71.7	67.9	71.7	73.8	71.5
	Thermometer. Range.	36.0	43.0	46.0	44.0	41.0	39.0	41.0	44.0	43.0	42.0
	Mean barometer.	29.976	29.996	29.990	30.034	29.999	29.997	29.942	29.984	29.999	29.991
	Mean humidity.	....	63.0	64.1	65.0	65.0	67.0	68.0	68.5	62.6	65.4
	Rainfall.	4.29	0.90	3.02	4.13	2.29	5.22	3.66	6.77	1.67	3.32
July.	Thermometer. Mean.	78.6	76.4	74.7	74.6	78.6	77.8	77.3	75.6	75.6	76.6
	Thermometer. Range.	28.0	33.0	31.0	31.0	40.0	34.0	33.0	35.0	35.0	33.4
	Mean barometer.	29.970	30.023	30.030	29.996	29.993	29.953	29.968	29.986	29 993	29.989
	Mean humidity.	....	62.7	64.1	66.0	63.0	70.0	67.0	65.6	65.2	65.4
	Rainfall.	9.20	5.00	2.25	3.63	5.71	5.53	4.35	3.62	7.74	5.22

TABLE B—*continued.*

	Meteorological elements.	1872	1873.	1874	1875	1876.	1877.	1878.	1879.	1880.	Mean of 9 years.
March	Thermometer. Mean.	75.9	72.3	71.1	72.4	74.3	75.5	73.4	72.7	72.8	73.4
	Thermometer. Range.	34.0	33.0	40.0	27.0	35.0	30.0	31.0	38.0	34.0	33.6
	Mean barometer	30.045	30.065	30.030	30.035	30.061	29.954	29.935	29.961	30.058	30.016
	Mean humidity.	....	75.4	61.3	76.0	69.0	69.0	70.0	73.2	70.2	70.5
	Rainfall.	7.81	11.49	5.65	6.42	0.98	0.66	3.83	7.13	5.09	5.45
August	Thermometer. Mean.	67.5	66.1	68.0	64.1	63.8	66.8	67.9	64.4	67.9	66.3
	Thermometer. Range.	40.0	43.0	36.0	45.0	42.0	36.0	44.0	43.0	41.0	40.9
	Mean barometer	30.049	30.096	30.100	30.035	29.984	30.077	30.153	30.144	30.064	30.078
	Mean humidity.	....	70.6	71.6	65.0	71.0	70.0	72.0	71.1	67.0	69.7
	Rainfall.	3.66	3.58	6.01	2.53	8.77	2.74	0.96	1.12	1.10	3.39
September	Thermometer. Mean.	52.9	54.7	55.5	53.7	54.8	57.4	58.1	61.7	55.1	56.0
	Thermometer. Range.	38.0	43.5	42.0	39.0	44.0	38.0	42.0	55.0	47.0	43.1
	Mean barometer.	30.105	30.073	30.115	30.015	30.020	30.062	30.053	30.168	30.123	30.082
	Mean humidity.	....	63.4	65.0	65.0	65.0	74.0	62.0	66.9	67.5	66.1
	Rainfall.	5.20	5.20	2.87	1.42	1.06	6.52	2.04	0.41	1.74	2.94
October.	Thermometer. Mean.	41.2	38.3	41.8	39.7	45.0	46.6	44.8	44.7	40.5	42.5
	Thermometer. Range.	42.0	35.0	45.0	54.0	54.0	46.0	34.0	56.0	59.0	47.2
	Mean barometer.	30.099	29.992	30.200	30.107	29.969	30.127	29.991	30.165	30.253	30.100
	Mean humidity.	....	63.9	61.0	68.0	71.0	72.0	66.0	68.0	67.9	67.2
	Rainfall.	3.40	5.10	2.32	5.40	7.31	5.14	2.19	1.38	1.75	3.78
November.	Thermometer. Mean.	28.9	38.3	35.7	34.7	25.5	40.6	34.5	38.8	28.6	34.0
	Thermometer. Range.	46.0	50.5	46.0	61.0	45.0	41.0	49.0	50.0	57.0	49.5
	Mean barometer.	30.160	30.165	30.176	30.042	30.027	30.179	30.018	30.154	30.048	30.107
	Mean humidity.	....	69.9	67.0	76.0	73.0	73.0	0.0	76.3	74.3	72.4
	Rainfall.	2.74	1.38	2.48	3.37	1.40	0.83	3.19	4.69	4.05	2.68
December.	Mean thermometer.	51.8	51.4	52.6	50.3	52.6	54.2	54.7	53.6	54.6	52.9
	Mean barometer.	30.047	30.041	30.089	30.059	30.037	30.048	29.987	30.073	30.092	30.053
	Mean humidity.	....	....	65.0	67.0	66.0	68.1	68.2	69.2	66.8	67.2
	Rainfall.	46.79	54.62	46.31	40.24	47.39	37.26	34.53	36.75	33.58	41.94
	No. of days on which rain fell.	....	160	136	154	131	113	118	128	122	133
	Prevailing winds.	N.W.	S. W.	S. W.	S. W.	W.	W.	S. W.	S. W.	S. W.	S. W.

The whole year.

F., June of 43° F., January of 44° F., October of 47° F., April of 52° F., February of 55° F., March, also, of 55° F., December of 57° F., and November of 59° F. This is nearly the usual sequence of the months as far as those of the summer and September are concerned, this season being the most equable of the year in Philadelphia; the absence of extremely cold weather in January, however, has given it an uncommonly high place in the list, while the warm spring has done a like thing for April; on the other hand, the premature approach of summer in May carried the range of this month much beyond its usual extent, and the early winter had a similar effect upon November. No frost was observed in the fall until the 3d of November.

Pressure.—The mean reading of the barometer for 1880 was 30.092 inches, which is .039 of an inch above the average for nine years, and the highest annual mean recorded during that period. In each of the months, with the exception of September and December, the mean monthly pressure was higher than the average, varying from an excess of .004 of an inch in July, to as much as .153 of an inch in November. The highest means are found, as a rule, in the winter months, and the lowest in the summer; but there was an exception this year as regards December, for notwithstanding the unusually low temperature of the month its mean barometric pressure was but 30.048 inches, exceeding only the respective means of April, June, and July.

The range of the barometer was through 1.612 inches, from 29.163 inches, the lowest point reached in February, to 30.775 inches, the highest recorded in

November. The former month was distinguished by the greatest barometric range, 1.474 inches, and July by the least, .854 of an inch. In general, the cold months were less equable as regards pressure than the warm ones.

Rainfall.—Only 33.58 inches of rain were recorded in 1880, or 8.36 inches less than the average precipitation during nine years; this is the least annual rainfall recorded in the Table which covers that period. The whole number of days on which rain or snow fell was 122, the average for eight years being 133. The lack in precipitation was divided, in unequal portions, among all the months save three, March, July, and December; the latter exceeded their averages, March very slightly, December 1.37 inches, and July as much as 2.52 inches, its rainfall of 7.74 inches being the highest recorded for any of the months. The least precipitation belonged to the exceptionally hot May, only .54 of an inch; from this value the months advanced as follows: September 1.10 inches, January 1.51 inches, June 1.67 inches, October 1.74 inches, November 1.75 inches, February and April each 2.43 inches, March 3.43 inches, December 4.05 inches, August 5.09 inches, and July, as noted above, 7.74 inches. The usual sequence of the months in this respect, and the average rainfall of each are as follows: May, September, and February each 2.52 inches, December 2.68 inches, January 2.93 inches, October 2.94 inches, March 3.31 inches, June 3.32 inches, September 3.39 inches, April 3.63 inches, November 3.78 inches, July 5.22 inches, and August 5.45 inches. Comparing the two sequences it will be seen that February, March, September, and December are

the farthest removed from their accustomed positions, changes due chiefly to a diminished rainfall in the autumn, and an increased precipitation in December; the last month, indeed, was visited by several heavy snow-storms, one being especially severe. It will be observed, further, if the months be combined into their respective seasons, that the spring, like the fall, was comparatively dry, while the summer was about as wet as usual.

Humidity.—The mean annual humidity for 1880 is expressed as 66.8°, saturation = 100°; this is a little less than the average for seven years, 67.2°. The month of highest mean humidity was January, its 75° being as much as 2.2° beyond its average, and that of lowest, April, with a mean humidity of 57.1°, or 2.5° below its average. Between these two the months came in the following order: December with a mean humidity of 74.3°, August of 70.2°, February of 68.4°, November of 67.9°, October of 67.5°, March of 67.3°, September of 67°, July of 65.2°, June of 62.6°, May of 59°. The greatest deviations from the accustomed values, besides those of January and April just mentioned, were found in June, September, and December, the first two months falling below their averages in degrees of humidity respectively 2.8° and 2.7°, and the latter going 1.9° beyond its mean.

Winds.—As usual in other years the prevailing direction of the wind was, in 1880, southwesterly. In September, November, and December, however, the wind blew chiefly from the northwest, and in February from that quarter and due west equally. In June the winds were, for the most part, from due west, while during January and March northeast

winds preponderated. In the latter month the wind blew with high velocity; in April there was a strong gale on the 30th day of the month.

Electricity.—Lightning was observed March 27th, and heavy thunder-storms were noted on the 4th and 7th days of April; also during May and July. In June, lightning was frequent; in August there were two auroras, on the 12th and 13th respectively, coinciding with hot and oppressive weather.

Epidemics.

The number of deaths returned by the Registration Office of the Board of Health of Philadelphia for the year 1880[1] was larger than that for any year since 1876. The whole number was 17,711, exclusive of the still-born (870), and those deaths recorded as from "premature births" (231). The mortality is greater by 2240 deaths than that of the previous year, and gives an annual death-rate for Philadelphia, with its population of 846,980 souls (U. S. census), of 20.91 deaths to each thousand of inhabitants living, or 2.26 deaths per thousand inhabitants more than in 1879; the latter year, however, as I showed in my last report,[2] was the healthiest to be found in the records of the Health Office since the registration of deaths had been efficiently carried out in this city. The rate of 1880, comparatively high though it may be, is still below the average death-rate for the previous ten

[1] Fifty-three weeks have been counted in 1880; from the week ending January 3, 1880, to that ending January 1, 1881, inclusive.

[2] Trans. Coll. Phys. Phila., 3d Series, Vol. V.

TABLE C.—COMPARISON OF CLASSIFIED LISTS OF CAUSES OF DEATH IN PHILADELPHIA DURING 1872, 1873, 1874, 1875, 1876, 1877, 1878, 1879 AND 1880.

Class.	Order.	Causes of death.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.
		All causes	18987	15224	15011	17805	18892	16004	15743	15471	17711
		Specified causes	18846	15087	14962	17752	18823	15982	15729	15461	17695
I.		Zymotic diseases	6299	3134	3005	4590	4777	3689	3303	2867	3810
II.		Constitutional diseases	3848	3616	3680	3880	4205	3924	3990	3844	4226
III.		Local diseases	6187	5859	6009	6803	7298	6115	6262	6422	7191
IV.		Developmental diseases	1935	1898	1662	1741	1943	1702	1658	1792	1794
V.		Violent deaths	577	589	606	648	600	552	516	536	673
		Causes not specified	141	137	49	53	69	22	14	10	17
			18987	15224	15011	17805	18892	16004	15743	15471	17711
I.	1	Miasmatic diseases	6175	3033	2903	4472	4638	3595	3210	2773	3693
	2	Enthetic diseases	24	33	40	24	34	43	34	37	24
	3	Dietic diseases	90	57	52	78	87	51	44	42	84
	4	Parasitic diseases	10	11	10	16	18	0	15	15	9
II.	1	Diathetic diseases	622	533	469	578	518	539	592	564	620
	2	Tubercular diseases	3226	3083	3211	3302	3687	3385	3398	3280	3606
III.	1	Dis. of the nervous system	2597	2370	2264	2619	2972	2277	2334	2219	2689
	2	" organs of circulation	776	715	771	835	869	829	847	898	909
	3	" respiratory organs	1651	1546	1688	1927	1927	1569	1598	1771	1907
	4	" digestive organs	838	922	907	1023	1056	976	961	1003	1137
	5	" urinary organs	242	237	273	367	370	346	399	410	452
	6	" generative organs	35	34	60	57	65	63	62	60	55
	7	" organs of locomotion	40	31	20	24	24	38	36	37	27
	8	" integumentary syst.	8	4	26	41	15	17	25	24	15
IV.	1	Develop. dis. of children	126	124	162	156	154	150	162	178	155
	2	" " adults	38	45	41	34	37	40	33	40	33
	3	" " old people	648	513	433	492	599	619	660	722	706
	4	Diseases of nutrition	1123	1216	1026	1059	1153	893	803	852	900
V.	1	Accident and negligence	513	521	528	553	518	476	462	462	587
	3	Homicide	17	14	19	27	22	[1]17	14	23	18
	4	Suicide	47	45	59	68	60	59	40	51	68
		Causes not specified	141	137	49	53	69	22	14	10	17
			18987	15224	15011	17805	18892	16004	15743	15471	17711
I.	1	Smallpox	2580	39	16	56	407	155	0	6	424
		Measles	140	29	117	12	53	69	12	8	108
		Scarlatina	168	309	352	1032	328	379	554	336	290
		Diphtheria	141	106	181	656	708	458	464	321	323
		Croup	284	188	196	429	386	338	388	291	303
		Whooping-cough	155	95	74	125	88	81	109	103	101
		Typhoid fever	341	358	468	400	774	542	404	344	495
		Typhus fever	35	31	26	21	27	15	9	1	23
		Erysipelas	76	89	83	87	84	77	67	83	63
		Puerperal fever	22	28	65	80	54	50	47	41	26
		Dysentery	75	73	49	78	78	79	51	60	98
		Diarrhœa	179	163	141	151	149	123	121	107	129
		Cholera infantum	1603	1068	859	992	1173	979	700	804	891
		Cholera morbus	111	67	51	47	35	43	33	27	73
		Malarial fevers	55	30	51	48	47	40	46	39	49
		Rheumatism	31	45	37	53	53	37	49	34	40
		Cerebro-spinal meningitis	125	238	80	83	85	56	90	62	78
		Other miasmatic diseases	54	77	57	122	109	74	66	106	159

[1] Includes four militiamen killed in the State service at Pittsburg.

TABLE C—*continued.*

Class.	Order	Causes of death.	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880
	2	Enthetic diseases	24	33	40	24	34	43	34	37	24
	3	Dietic diseases	90	57	52	78	87	51	44	42	84
	4	Parasitic diseases	10	11	10	16	18	0	15	15	9
II.	1	Cancer	334	261	306	319	302	327	380	363	369
		Other diathetic diseases	280	272	163	259	216	212	212	201	251
	2	Tabes mesenterica	779	679	721	766	811	786	738	649	737
		Phthisis pulmonalis	2248	2228	2277	2359	2676	2411	2491	2481	2692
		Other tubercular diseases	199	176	213	177	200	188	169	150	177
III.	1	Cephalitis	956	874	747	935	1023	724	729	703	424
		Apoplexy	260	269	232	260	279	199	267	268	303
		Paralysis	236	233	249	287	305	309	323	317	385
		Convulsions	732	670	675	811	894	703	694	626	737
		Sunstroke	135	16	17	20	130	8	28	3	37
		Other dis. of nervous system	278	308	344	306	341	334	293	302	303
	2	Pericarditis	45	49	87	87	106	87	99	124	127
		Aneurism	9	21	18	23	20	27	24	25	35
		Heart diseases	644	582	626	670	701	676	695	704	728
		Other dis. organs of circulat'n	78	63	40	55	42	39	27	45	19
	3	Bronchitis	214	206	256	342	354	307	326	301	414
		Pleurisy	50	65	82	104	73	70	54	67	46
		Pneumonia	1252	1132	1133	1277	1306	1063	1020	1208	1253
		Other dis. of respirat'y organs	135	143	217	204	194	129	198	195	194
	4	Enteritis	306	337	336	399	431	356	361	340	463
		Peritonitis	131	181	119	159	130	143	158	194	177
		Intestine diseases	133	111	120	150	133	133	129	122	134
		Stomach diseases	6	23	37	29	32	27	21	33	26
		Liver diseases	227	257	227	219	271	247	224	271	288
		Other dis. of digestive organs	35	13	68	67	59	70	68	43	49
	5	Nephritis	17	19	24	47	35	30	29	35	52
		Bright's disease	36	29	131	195	263	204	224	253	256
		Other dis. of urinary organs	189	189	118	125	72	112	146	122	144
	6	Diseases of ovaries	6	8	16	13	8	14	22	18	20
		Uterus diseases	29	26	44	44	57	49	40	42	35
	7	Caries and necrosis	31	22	10	12	13	25	25	26	18
		Joint diseases	9	8	10	12	11	13	11	11	9
	8	Dis. of integumentary system	8	4	26	41	15	17	25	24	15
IV.	1	Malformations	88	90	123	120	126	120	132	144	126
		Teething	38	34	39	36	28	30	30	34	29
	2	Childbirth	10	15	16	10	8	12	10	16	8
		Puerperal convulsions	28	27	23	24	23	28	13	18	19
		Other develop. dis. of adults	0	3	2	0	6	0	10	6	6
	3	Old age	648	513	433	492	599	619	660	722	706
	4	Debility	[1]822	[1]921	712	695	681	530	448	472	520
		Inanition	301	295	314	364	472	363	355	380	380
V.	1	Drowning	99	112	129	119	111	127	129	105	115
		Otherwise	414	409	399	434	407	349	333	357	472
	3	Homicide	17	14	19	27	22	17	14	23	18
	4	Suicide	47	45	59	68	60	59	40	51	68
		Causes not specified	141	137	49	53	69	22	14	10	17
			18987	15224	15011	17805	18892	16004	15743	15471	17711

[1] Deaths from "premature birth" are included.

NOTE.—In the table the number of deaths from "premature births" and of the "still-born" have been omitted. They were recorded as follows:—

	1872.	1873.	1874.	1875.	1876.	1877.	1878.	1879.	1880.
Premature births	...	...	229	280	244	261	262	190	231
Still-born	825	885	891	824	896	930	908	811	870

years (1870-1879), which I have calculated on the basis of the United States census of 1870 and that for 1880—assuming the increase of the population during the decade to have been in equal increments for each succeeding year—to be 21.65 deaths to each thousand inhabitants living. The mortality is, also, as it is wont to be, below, and, in some instances, much below that in the several American and foreign cities for 1880, the death-rates of which are as follows: New York 26.31 deaths per thousand inhabitants, Brooklyn 23.33, Boston 23.7, Baltimore 24.32, London 22.2, Paris 29.0, Berlin 29.9, Vienna 27.2, Amsterdam 27.2, Copenhagen 25.0, Stockholm 28.8, St. Petersburg 46.1, Madrid 40.1, Rome 32.0.[1]

In my report for 1879, I said that the low mortality of that year might have been anticipated from its meteorological conditions; in 1880, however, the climatic elements did not give a like favorable augury. The mildness of the early months, it is true, promised fewer deaths from diseases of the respiratory organs, but the early advent of hot weather in May, and its continuance into September, by adding to the length of the summer, the most unhealthy season of our year, gave grounds for apprehension that such reduction would be more than counterbalanced by the summer mortality, while the excessive cold of December foretold for that month more deaths from those causes which are most destructive in winter.

Of the 17,711 deaths reported, 9075 were of males, and the lesser number, 8636, of females. They were

[1] The death-rates of the foreign cities are taken from the annual summary of births, deaths, and causes of death in London, etc., 1880. London, 1881.

divided among the several ages as follows: under one year of age 4003, or 22.6 per cent. of the total mortality; between one and two years 1285, or 7.26 per cent.; between two and five years 1306, or 7.37 per cent.; between five and ten years 671, or 3.79 per cent.; between ten and fifteen years 322, or 1.82 per cent.; between fifteen and twenty years 503, or 2.84 per cent.; between twenty and thirty years 1834, or 10.36 per cent.; between thirty and forty years 1657, or 9.36 per cent.; between forty and fifty years 1474, or 8.32 per cent.; between fifty and sixty years 1356, or 7.66 per cent.; between sixty and seventy years 1360, or 7.67 per cent.; between seventy and eighty years 1174, or 6.65 per cent.; and beyond eighty years 766, or 4.32 per cent. The percentages above mark, as usual, a greater mortality in the first years of life; as many as 37.23 per cent. of the whole number of deaths occurring in children under five years of age. In the previous exceptionally healthy year, the proportion was 35.69 per cent., a percentage not very much less, so that the greater mortality of 1880 did not fall exclusively upon the very young.

In the classification of the deaths according to their causes (Table C), 3810 of the whole number 17,711, or 21.51 per cent., are found in the class of ZYMOTIC DISEASES; 4226, or 23.86 per cent., in that of CONSTITUTIONAL DISEASES; 7191, or 40.60 per cent., in that of LOCAL DISEASES; 1794, or 10.13 per cent., in that of DEVELOPMENTAL DISEASES; and 673, or 3.80 per cent., in that of VIOLENT DEATHS; while 17, or one-tenth of 1 per cent., are under the caption CAUSES NOT SPECIFIED, which last is made to include those cases in which the causes of death were too ill-defined

TABLE D.—A CLASSIFIED LIST OF THE NUMBER OF DEATHS FROM EACH CAUSE IN PHILADELPHIA DURING EACH MONTH OF 1880, AND DURING THE WHOLE YEAR (53 WEEKS, DECEMBER 28, 1879, TO JANUARY 1, 1881).

Class.	Order.	Causes of death.	January (5 weeks).	February (4 weeks).	March (5 weeks).	April (4 weeks).	May (4 weeks).	June (5 weeks).	July (4 weeks).	August (4 weeks).	September (5 weeks).	October (4 weeks).	November (4 weeks).	December (5 weeks).	The whole year (53 weeks.)
		All causes	1465	1211	1689	1354	1308	2011	1741	1304	1406	1150	1252	1820	17711
		Specified causes	1463	1210	1689	1354	1304	2010	1741	1302	1402	1147	1251	1820	17705
I		Zymotic diseases	227	187	258	194	220	505	558	332	280	224	289	475	3850
II		Constitutional diseases	304	248	420	320	339	428	395	323	360	299	288	392	4226
III		Local diseases	675	537	795	631	546	747	566	457	540	451	508	748	7201
IV		Developmental diseases	150	147	169	133	140	200	161	129	148	117	121	159	1774
V		Violent deaths	47	41	47	56	60	69	61	61	74	56	45	56	673
		Causes not specified	2	1	0	0	3	1	0	2	4	3	1	0	17
			1465	1211	1689	1354	1308	2011	1741	1304	1406	1150	1252	1820	17711
I	1	Miasmatic diseases	215	183	247	188	211	558	551	326	264	213	276	461	3693
	2	Enthetic diseases	4	2	3	1	0	1	1	2	3	0	4	3	24
	3	Dietic diseases	8	2	8	3	9	6	5	4	11	8	9	11	84
	4	Parasitic diseases	0	0	0	2	0	1	1	0	2	3	0	0	9
II	1	Death-like diseases	62	33	56	53	47	69	57	35	53	51	40	64	620
	2	Tubercular diseases	302	265	364	267	292	359	338	288	307	248	248	328	3606
III	1	Dis. of the nervous system	224	168	258	219	204	341	258	205	252	161	178	241	2569
	2	" organs of circulation	94	73	96	72	66	69	62	53	66	70	70	115	806
	3	" respiratory organs	215	193	300	220	154	117	86	59	79	12	151	213	1907
	4	" digestive organs	85	69	103	71	91	157	120	103	94	56	66	122	1137
	5	" urinary organs	44	28	33	40	24	58	38	31	40	36	35	45	452
	6	" generative organs	3	3	3	6	6	8	0	2	5	6	6	7	55
	7	" organs of locomotion	4	0	2	3	1	5	1	2	3	1	1	4	27
	8	" integumentary syst.	1	3	0	0	0	2	1	2	1	1	1	3	15
IV.	1	Develop. dis. of children	13	15	21	10	12	18	9	8	17	7	14	11	155
	2	" " adults	2	3	4	4	1	4	3	1	5	2	2	2	33
	3	" " old people	73	61	67	64	58	69	49	42	47	50	49	77	706
	4	Diseases of nutrition	62	68	77	75	69	109	100	78	79	58	56	69	900
V.	1	Accident and negligence	40	33	41	48	47	59	54	57	69	49	41	49	587
	3	Homicide	2	3	1	2	3	1	1	2	0	2	0	1	18
	4	Suicide	5	5	5	6	10	9	6	2	5	5	4	6	68
		Causes not specified	2	1	0	0	3	1	0	2	4	3	1	0	17
			1465	1211	1689	1354	1308	2011	1741	1304	1406	1150	1252	1820	17711
I	1	Smallpox	13	15	8	5	9	7	4	13	33	43	85	189	424
		Measles	5	20	38	29	9	4	0	0	0	1	0	2	108
		Scarlatina	17	28	11	17	32	39	11	20	17	16	32	50	290
		Diphtheria	35	30	34	18	25	29	15	8	28	59	40	31	323
		Croup	40	28	39	24	22	16	16	10	16	24	23	45	293
		Whooping-cough	10	3	14	3	12	11	13	7	6	8	3	11	101
		Typhoid fever	39	29	35	27	30	25	40	65	46	38	49	72	495
		Typhus fever	1	1	4	4	1	0	3	0	2	1	1	5	25
		Erysipelas	9	8	12	6	5	7	0	2	2	3	2	7	63
		Puerperal fever	4	1	5	5	2	3	1	1	0	0	2	2	26
		Dysentery	0	1	3	7	4	8	26	24	14	5	4	2	98
		Diarrhœa	6	1	10	9	6	15	24	20	14	9	7	8	129
		Cholera infantum	3	0	3	8	13	328	342	130	56	4	0	4	891
		Cholera morbus	2	0	0	0	1	21	33	10	3	2	1	0	73
		Malarial fevers	0	2	3	3	1	2	9	3	9	8	4	5	49
		Rheumatism	7	2	3	5	4	15	4	1	2	6	5	6	60
		Cerebro-spinal meningitis	6	3	9	7	11	9	2	3	6	4	6	12	78
		Other miasmatic diseases	18	11	16	11	24	19	8	9	10	11	12	10	159
	2	Enthetic diseases	4	2	3	1	0	1	1	2	3	0	4	3	24
	3	Dietic diseases	8	2	8	3	9	6	5	4	11	8	9	11	84
	4	Parasitic diseases	0	0	0	2	0	1	1	0	2	3	0	0	9

TABLE D—*continued.*

Class.	Order	Causes of death	January (5 weeks).	February (4 weeks).	March (5 weeks).	April (4 weeks).	May (4 weeks).	June (5 weeks	July (4 weeks).	August (4 weeks).	September (5 weeks).	October (4 weeks	November (4 weeks	December (5 weeks).	The whole year (52 weeks	
II	1	Cancer	43	17	32	32	26	47	31	20	30	32	23	36		9
		Other diathetic diseases	19	19	24	21	21	22	26	15	23	19	17	28		24
	2	Tabes mesenterica	39	38	46	51	44	103	130	100	89	48	35	14		737
		Phthisis pulmonalis	248	217	302	197	232	238	187	176	206	197	203	289		2492
		Other tubercular diseases	15	10	16	19	16	18	21	12	12	3	10	25		177
III	1	Cephalitis	78	62	80	71	71	117	84	78	86	57	59	81		834
		Apoplexy	25	21	26	19	23	26	19	18	32	29	35	38		363
		Paralysis	38	19	41	39	24	30	27	33	38	18	33	10		85
		Convulsions	66	48	83	62	59	108	69	51	67	37	34	53		757
		Sunstroke	0	0	0	0	5	18	12	0	2	0	0	0		37
		Other dis. of nervous system	22	18	28	28	22	32	47	25	27	20	17	17		303
	2	Pericarditis	17	8	10	13	11	9	8	7	8	8	12	16		127
		Aneurism	4	4	3	4	2	2	1	4	3	1	5	2		35
		Heart diseases	78	60	83	54	53	57	52	39	50	60	51	91		728
		Other dis. organs of circulation	0	1	0	1	0	1	1	3	5	1	2	4		19
	3	Bronchitis	46	31	66	44	35	20	25	15	16	31	43	42		414
		Pleurisy	1	1	6	5	1	10	2	2	3	2	4	4		46
		Pneumonia	148	140	201	155	97	68	47	37	47	74	95	144		1363
		Other dis. of respiratory organs	20	21	27	16	21	19	12	5	13	13	9	18		204
	4	Enteritis	33	22	34	28	31	72	60	58	39	22	22	42		463
		Peritonitis	13	14	24	14	22	26	14	9	14	5	5	17		177
		Intestinal diseases	9	14	14	2	10	17	17	9	8	9	8	17		134
		Stomach diseases	5	1	5	0	0	5	1	1	0	1	1	0		25
		Liver diseases	24	15	24	24	24	30	26	25	28	16	22	30		288
		Other dis. of digestive organs	1	3	2	3	4	7	2	1	5	3	8	10		49
	5	Nephritis	6	3	5	4	4	6	12	3	2	3	1	3		52
		Bright's disease	28	15	14	24	16	34	13	14	25	24	22	27		276
		Other dis. of urinary organs	10	10	14	12	4	18	13	14	13	9	12	15		144
	6	Diseases of ovaries	0	0	2	4	4	3	0	0	2	1	1	3		20
		Uterus diseases	3	3	1	2	2	5	0	2	3	5	5	4		35
	7	Caries and necrosis	2	0	0	3	1	4	0	1	2	1	1	3		18
		Joint diseases	2	0	2	0	0	1	1	1	1	0	0	1		9
	8	Dis. of integumentary system	1	3	0	0	0	2	1	2	1	1	1	3		15
IV	1	Malformations	12	11	18	9	9	15	3	4	14	7	14	10		126
		Teething	1	4	3	1	3	3	6	4	3	0	0	1		29
	2	Childbirth	0	0	3	0	1	2	1	0	1	0	0	0		8
		Puerperal convulsions	2	1	1	1	0	1	2	1	4	2	2	2		19
		Other develop. dis. of adults	0	2	0	3	0	1	0	0	0	0	0	0		6
	3	Old age	73	61	67	64	58	69	49	42	47	50	49	77		706
	4	Debility	40	38	51	38	46	62	57	37	42	42	29	38		500
		Inanition	22	30	26	37	23	47	43	41	37	16	27	31		389
V.	1	Drowning	3	3	4	11	11	22	25	10	16	5	4	1		115
		Otherwise	37	30	37	37	36	37	29	47	53	44	37	48		472
	3	Homicide	2	3	1	2	3	1	1	2	0	2	0	1		18
	4	Suicide	5	5	5	6	10	9	6	2	5	5	4	6		68
		Causes not specified	2	1	0	0	3	1	0	2	4	3	1	0		17
			1465	1211	1689	1354	1308	2011	1741	1504	1406	1150	1252	1820		17711

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to admit of proper classification. The percentage of the total mortality belonging to deaths from zymotic diseases in the previous year was 18.53, or about 2 per cent. of the mortality of 1880 less than in the latter year: on the other hand, while constitutional diseases caused 24.85 per cent. of the whole number of deaths in 1879, this class of maladies were in 1880 responsible for one per cent. less of the total mortality, though, on account of the large increase in the number of deaths from all causes in 1880, their actual mortality exceeded that from the same diseases in 1879; the proportion of deaths in 1879 from local diseases, 41.51 per cent., and that from developmental diseases, 11.58 per cent., are in each instance greater than fell to these classes in 1880, though, as with constitutional diseases, the real number of deaths from these diseases was larger in 1880 than in 1879; the percentage of mortality due to violent deaths in the earlier year, 3.46 per cent., was somewhat less than that calculated for the same class in 1880.

When we compare the actual number of deaths from zymotic diseases in the two years we find the excess in 1880 to be 943, or about one-third of the mortality in 1879. This increase was due especially to the prevalence of smallpox; measles also caused some deaths, and the mortality from typhoid fever was markedly aggravated; there were also more deaths from cholera infantum; on the other hand, scarlatina diminished the number of its victims, while diphtheria, croup, and whooping cough each claimed for itself about the same number of deaths in the two years.

The reappearance of *smallpox* among the causes of

death at the close of the year 1879, was followed by a mortality of 424 in 1880. The whole number of cases of the disease reported to the Health Office during the year being 1832,[1] it would follow that the mortality was one death in 4.32 cases. There were, however, undoubtedly many light cases of variolous disease which failed to reach the Health Office, and even some severe ones, since it is not very difficult to hide a case when death does not occur, so that it is probable that the disease was not so fatal as it appears. According to the returns, the ratio of the deaths to cases varied very much in the several wards, for while there were 12 cases reported in the Ninth ward with no deaths, 11 in the Thirteenth, also without a death, and 17 cases with but 1 death in the Thirtieth, there was in the Seventeenth and Twenty-eighth wards 1 death to about every 4 cases reported.

With the exception of the Ninth and Thirteenth wards, already mentioned, there were deaths from smallpox in each of the wards of the city (Table E); but the Twelfth, Twenty-seventh, and Thirtieth had each only one death from the disease, and the Eighth, Eleventh, and Twenty-first each only two. The most deaths were reported from the Twenty-eighth ward, 119; but these included the deaths in the Municipal Hospital for Contagious Diseases, to which cases of smallpox from all parts of the city were taken; excluding these, 79 in number, the deaths really belonging to the ward are, however, as many as 40. In the Second ward 46 persons died from the disease, in the Fourth 34, in the Thirty-first 31, in the Nineteenth

[1] Health Office's Annual Report, etc., 1880, p. 172. Philadelphia, 1881.

TABLE E.—DISTRIBUTION OF THE DEATHS FROM THE ZYMOTIC DISEASES SMALLPOX, SCARLATINA, DIPHTHERIA, CROUP, AND TYPHOID FEVER, AMONG THE SEVERAL WARDS OF PHILADELPHIA IN 1880, DURING EACH MONTH AND FOR THE YEAR.

Number of ward.	January.					February.					March.					April.					May					June					July.					August				
	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typhoid fev.	Smallpox.	Scarl. fina.	Diphtheria	Croup.	Typhoid fev.	Smallpox.	Scarlatina.	Diphtheria	Croup.	Typhoid fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typhoid fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typhoid fev.	Smallpox	Scarlatina.	Diphtheria.	Croup.	Typhoid fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typhoid fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typhoid fev.
1st	..	1	1	4	1	..	1	..	..	..	..	1	..	2	3	..	1	2	1	..	..	1	2	..	2	..	6	1	..	2	..	..	..	..	1	1	6
2d	..	..	..	3	1	..	..	..	2	2	..	1	2	1	..	1	1	..	..	..	..	1	..	1	3	..	1	..	..	3	..	..	..	2	..	1	..	5
3d	..	..	..	1	..	..	..	..	..	..	..	..	3	..	..	..	..	..	..	..	2	..	..	..	3	..	3	..	..	..	..	..	1	2	..	1	..	..	2
4th	..	..	..	1	..	..	1	..	1	2	..	1	2	..	..	..	1	..	..	..	1	..	1	..	..	..	1	..	..	..	..	..	..	..	7	1	1	..	1
5th	..	..	1	..	..	..	..	1	1	..	1	1	..	..	..	..	..	..	..	..	2	..	..	..	..	1	1	..	1	..	..	..	..	4	..	..	..	1	2
6th	..	..	1	..	..	..	..	1	..	..	1	2	..	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	..	..	..	..	1
7th	..	..	1	5	..	..	..	1	..	2	..	..	4	..	..	..	2	2	1	..	1	..	..	1	..	..	..	2	..	..	2	..	..	..	2	..	..	..	1
8th	..	..	..	1	..	..	..	..	1	..	..	..	1	..	..	..	1	1	1	..	..	..	..	1	..	..	1	..	2	..	..	..	2	..	1	..	..	1	2
9th	..	..	..	..	1	..	2	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	1	..	..	..	2	..	..	..	1	2	..	1	..	..	..	..
10th	..	..	..	1	..	..	2	2	1	..	..	..	..	..	..	2	..	..	1	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1
11th	..	..	..	..	..	..	1	..	1	..	..	..	..	1	..	1	2	..	..	..	1	1	..	..	..	..	2	..	1	..	..	..	..	..	..	..	..	..	3
12th	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..	..	..	..	..	..	1	..	..	..	..	..	2	..	..	..	..	..	2
13th	..	..	..	..	3	..	1	..	..	1	..	..	1	1	2	..	..	1	2	..	1	2	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
14th	1	..	1	2	1	1	1	3	1	1	..	..	3	..	1	..	..	..	..	..	..	..	..	1	..	..	1	..	..	1	..	..	..	2	..	..	..	..	1
15th	1	1	3	..	2	..	1	..	2	2	..	1	2	1	..	..	..	..	1	..	1	3	2	..	..	..	1	..	3	1	1	..	..	..	2	1	..	..	6
16th	..	..	1	1	3	..	..	..	..	1	..	..	1	1	2	..	..	..	1	..	..	1	..	..	..	..	2	..	1	1	..	1	..	2	..	1	..	..	..
17th	..	..	1	1	2	..	..	1	..	..	..	..	..	1	1	..	2	1	..	..	..	1	1	..	1	..	2	4	4	..	..	..	1	2	1	..	3	..	2	1
18th	..	1	3	2	1	..	3	3	1	4	..	..	2	2	1	..	1	..	3	3	..	1	2	3	..	..	1	3	1	1	..	..	1	..	1	..	2	2	3
19th	1	1	2	3	1	2	4	1	1	1	2	2	..	3	1	2	..	1	2	4	2	4	..	1	4	2	2	1	3	1	..	4	2	2	2	..	2	1	..	4
20th	..	1	..	4	..	..	1	4	5	2	..	1	1	1	1	..	4	1	1	2	..	3	..	1	3	..	..	1	..	..	..	1	1	1	..	..	..	1	3
21st	..	1	2	1	2	..	..	1	..	..	..	..	1	2	2	..	..	..	..	..	..	..	..	1	..	..	4	2	1	..	..	..	1	1	..	..	1	..	..	..
22d	..	1	1	..	1	..	..	1	..	..	..	..	1	2	2	..	..	..	..	1	..	..	1	..	1	..	5	..	..	..	..	2	..	..	2	..	2	..	5
23d	..	..	4	..	1	..	..	1	..	2	1	1	..	1	1	..	..	1	..	3	1	4	4	2	..	..	4	1	..	2	..	..	1	..	2	..	3	..	..	1
24th	..	2	2	..	7	..	3	2	..	1	..	2	1	1	2	..	..	..	1	..	3	1	3	..	..	..	1	1	1	..	1	..	1	..	2	..	..	..	1
25th	2	..	7	5	3	1	3	2	1	..	1	..	1	..	2	2	2	2	..	3	2	1	1	2	2	..	..	1	..	1	1	..	2	..	2	..	1	2	..	1
26th	..	2	1	1	1	..	1	1	1	2	..	..	2	3	2	..	..	..	3	..	..	1	..	1	1	..	1	1	1	1	..	..	..	..	1	..	1	..	..
27th	..	..	..	..	..	..	..	..	..	2	..	..	..	1	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	5
28th	4	4	..	3	2	5	1	2	1	..	1	..	3	3	..	1	..	..	2	..	2	2	1	..	..	4	..	..	1	..	1	..	..	..	3	..	..	..	3
29th	1	..	..	1	2	1	1	3	4	3	1	2	1	2	4	..	1	3	2	1	..	..	..	..	1	..	2	1	..	..	..	1	2	3	..	1	..	..	3
30th	..	..	1	..	1	..	1	..	1	1	..	..	..	..	3	..	..	..	1	1	..	..	1	2	..	..	..	..	1	..	..	..	1	2	..	..	..	..	2
31st	3	2	1	..	3	5	..	..	3	..	2	..	5	1	2	..	..	1	1	2	1	3	2	2	1	..	2	1	4	..	1	..	4	2	..	1	..	..	..
Total	13	17	35	40	39	15	28	30	28	29	8	11	31	39	35	5	17	18	24	27	9	32	25	22	31	7	39	29	16	24	4	11	15	16	40	13	20	8	10	65

TABLE E—*continued.*

Number of ward.	September.					October.					November.					December.					For the year.					Population, census of 1880.	Area in square miles.
	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.		
1st	1	..	1	1	3	2	..	..	2	2	7	1	2	1	1	11	3	3	3	2	21	15	13	15	23	43,085	5.509
2d	5	..	..	2	3	14	..	..	1	..	16	1	2	..	..	11	1	3	1	6	46	6	7	13	26	28,498	.442
3d	..	2	..	..	..	3	1	..	..	..	5	..	1	2	..	6	..	..	..	1	14	9	1	7	8	18,271	.191
4th	8	2	1	..	3	7	1	1	1	1	8	4	..	..	1	4	2	..	1	..	34	13	5	7	8	18,853	.229
5th	..	..	..	..	..	..	..	1	..	..	3	2	1	1	1	7	1	..	2	2	10	7	6	5	10	16,368	.321
6th	..	..	..	..	..	..	..	..	..	1	2	1	..	..	1	1	..	..	..	..	3	2	5	4	4	10,004	.321
7th	1	..	1	..	1	1	..	..	..	..	1	..	1	1	3	5	..	1	1	2	11	1	13	9	12	31,087	.439
8th	1	..	..	..	1	..	..	..	..	1	..	..	..	..	..	1	..	..	2	..	2	1	4	6	9	19,545	.435
9th	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	0	5	4	1	3	12,481	.400
10th	1	3	2	1	2	..	1	..	..	..	..	..	1	..	..	4	2	2	..	2	6	10	9	3	6	23,363	.359
11th	..	1	1	..	..	..	3	1	..	1	1	1	..	..	1	1	2	..	2	..	2	12	5	4	6	12,930	.210
12th	..	..	..	..	..	..	..	1	..	1	2	..	..	..	1	..	1	..	..	1	1	3	2	2	8	14,690	.193
13th	..	..	3	..	..	..	..	..	..	..	1	..	1	1	1	3	..	..	..	..	4	0	3	7	3	17	18,646	.259
14th	2	..	..	..	..	..	..	..	..	..	2	..	..	1	1	1	3	1	..	..	4	7	3	8	4	15	22,354	.300
15th	..	..	4	..	6	..	..	1	3	1	1	1	5	..	2	2	..	1	2	2	4	5	22	14	23	47,865	1.049
16th	1	..	1	..	1	..	..	1	1	1	..	2	..	..	2	4	..	..	4	3	5	6	5	9	16	17,802	.281
17th	..	..	..	3	2	..	1	..	1	1	2	1	2	..	4	8	3	..	2	2	10	13	11	16	15	20,451	.251
18th	..	..	..	1	1	..	1	1	2	2	1	..	1	2	2	5	3	1	4	2	6	13	17	23	21	29,354	.650
19th	..	2	2	1	1	1	1	2	1	2	4	..	1	1	4	11	4	2	3	7	27	26	15	23	32	43,887	.698
20th	1	1	1	1	4	..	1	2	2	2	1	4	2	2	5	4	4	1	..	8	6	20	14	19	31	43,207	.734
21st	..	..	..	1	3	..	1	1	1	3	1	2	..	..	1	1	5	..	1	1	2	15	8	7	13	19,699	7.129
22d	..	..	7	..	4	1	1	9	1	2	..	..	6	1	1	3	2	5	..	..	4	13	33	4	21	31,798	18.114
23d	..	3	2	..	1	..	..	3	1	2	2	3	1	..	..	..	8	2	..	3	4	26	20	4	16	26,522	42.716
24th	..	..	..	..	1	..	..	..	3	1	..	..	1	3	5	5	..	1	2	4	5	11	9	13	26	46,057	9.725
25th	..	2	1	2	1	..	1	2	..	3	1	3	6	3	1	10	2	..	1	1	20	15	27	14	20	36,104	10.359
26th	..	..	..	1	1	2	2	..	2	1	5	..	..	..	..	2	3	..	3	5	3	10	7	9	18	15	35,138	7.481
27th	..	..	1	..	1	..	..	..	..	..	1	..	..	2	..	..	..	..	..	1	..	1	0	1	6	10	23,284	11 680
28th	11	..	..	2	2	10	..	4	..	1	17	2	2	1	2	60	1	5	..	2	119*	10	17	13	12	34,442	6 343
29th	..	..	..	..	2	1	..	..	..	2	3	1	2	..	1	5	1	..	1	3	12	9	11	12	25	40,787	1.409
30th	1	..	..	..	2	..	..	1	..	2	..	..	..	..	1	..	1	1	4	5	1	2	3	8	23	29,100	.549
31st	..	1	..	..	..	1	..	..	1	2	3	..	1	1	3	14	3	..	3	2	31	9	12	17	21	31,308	.713
Total	33	17	28	16	46	43	16	30	24	38	85	32	40	23	49	189	50	31	45	72	424	290	323	303	495	846,980	129.450

* The Smallpox Hospital is situated in the 28th ward.

27, in the First 21, in the Twenty-fifth 20, the Third 14, and the Seventh 11. Each of the remaining wards had 10 or a less number of deaths. If we group the wards together, we will find that in the southern section of the city—all that part below South Street and East of Broad Street, comprising the First, Second, Third, and Fourth wards—there were, in a population of 108,707 souls, deaths from smallpox to the number of 115, or a death-rate from the disease of 10.5 to the 10,000 inhabitants; all these deaths took place in and after the month of August. In another group of wards in the northeastern quarter of the city, comprising the ward in which smallpox first appeared in the previous year, namely, the Twenty-fifth, the Nineteenth, the Thirty-first, and the Twenty-eighth, excluding the deaths at the smallpox hospital, there were 118 deaths among a population of 145,741, or a death-rate of 8.10 per each 10,000; in this district the deaths were distributed throughout the whole year, though the summer was nearly exempt. These two groups of wards are the same in which the smallpox raged most fiercely in the great epidemic of 1871–72, and again in the lesser one of 1876. Between these two districts stretches the part of the city where the population becomes densest, the greatest movement of business is to be found, and the wealthier classes have their homes (the Fifth, Sixth, Seventh, Eighth, Ninth, Tenth, Eleventh, Twelfth, Thirteenth, Fourteenth, Fifteenth, Sixteenth, Seventeenth, Eighteenth, Twentieth, and Twenty-ninth wards); here, in a population of 380,934, there were but 85 deaths, or 2.2 to each 10,000 of inhabitants.

It may seem at first sight singular that the disease

passed comparatively harmlessly over the latter region to establish itself in the southern wards with more virulence than it displayed in its birthplace, but no doubt the explanation is to be found in the greater attention paid to vaccination and cleanliness in the richer quarter of the town. In the outer circle of wards, Twenty-first (Manayunk), Twenty-second (Germantown), Twenty-third (Frankford and Bridesburg), Twenty-fourth and Twenty-seventh (West Philadelphia), Twenty-sixth and Thirtieth, the aggregate population of which is 165,206 souls, there were but 27 deaths, or 1.6 to each 10,000 of inhabitants. The distance of these districts from the focus of the disease suggests itself as the cause of their comparative exemption.

Of the deaths from smallpox, 36 were recorded for the first quarter of the year, 21 for the second, 50 for the third, and as many as 317 for the last quarter; the figures show the insidious advance of the disease, not many deaths during the first three months, then a decline in the number in the second quarter, during which the lowest monthly mortality, 4, was reached; in the third quarter a rise to a number of deaths almost equal to that recorded for the whole first half of the year, and finally in the last quarter a mortality nearly three times as great as that returned for all the preceding quarters of the year, the closing month, December, claiming the maximum monthly mortality, 189. A tendency in smallpox to a diminished mortality or disappearance in the summer, and to a beginning or increase in the number of cases in the later months of the year has been shown in Philadelphia during and since the severe epidemic of 1871–

1872; before that event registration was not full enough to leave data to elucidate these points. In the epidemic mentioned the deaths from the disease began to increase in the early autumn; there was a marked rise in the mortality during the first week in October, after which the progress of the disease was very rapid; the winter gave a high mortality, but by the time the summer came the scourge had nearly disappeared, and there was no rekindling of the dying flame in that fall or the succeeding winter, the disease entirely vanishing from the causes of death in the following summer. Subsequently there was very little smallpox in Philadelphia till the fall of 1875, when a sharp increase in the number of its deaths took place, followed by a small epidemic in 1876, which diminished, however, in mortality in the summer, to increase a little in the fall of that year, and finally to disappear entirely in the summer of 1877; after which the mortuary lists were free from smallpox until the reappearance of the disease in the fall of 1879. Briefly to recapitulate, we have the great epidemic of 1871–1872, beginning in the fall of 1871, the much smaller one of 1876 in the autumn of 1875, again a new appearance of the disease in the last months of 1879, and a large increase in its mortality during the autumn of 1880, after the deaths had diminished to a very few during the summer of that year. The Registrar-General of England, in his "annual summary"[1] for 1880, calls attention to a like seasonal distribution of the deaths from smallpox in London, the observations extending over five years;

[1] Ibid., pp. x–xi.

but such distribution has not been generally noted in other places. It is perhaps to be accounted for in the same way that we explain the greater prevalence of typhus in winter, by the greater deficiency of ventilation in houses, and the keeping within doors at that season: conditions favorable to the accumulation and reception of the poison of the disease.

Speaking in my report for 1879 of smallpox, I detailed the measures of isolation, vaccination, re-vaccination, and purification of surroundings instituted by the health authorities of the city to oppose the march of the disease, and there can be no doubt that these were effective in staying its ravages; but these authorities were armed with no law to compel obedience to their mandates, and in a few months even the money appropriated to carry them out was exhausted. Then their hands were to a certain extent tied—not entirely, for vaccination and the removal of nuisances still went on, but only by the good-will of the vaccine physicians, and as regards the purification of places by more indirect methods of the health officer. Finally, after vexatious delays, more money was secured for vaccination, and the work went on more briskly. It would take, indeed, what seems to legislators an immense amount of money for such a purpose to stamp out smallpox in so large a city as Philadelphia. According to Curschman,[1] the duration of immunity from the disease under exposure after vaccination may be stated at from eight to ten or twelve years. If we put it at ten years, there would then be required in our popula-

[1] Encyclopædia of the Practice of Medicine; Ziemssen, vol. ii. p. 406. Am. Edition. New York: Wm. Wood & Co., 1875.

tion of, in round numbers, 850,000 inhabitants, a yearly average of 85,000 vaccinations and re-vaccinations to keep the city protected; that is, one-tenth of the whole population would need to have the operation performed upon it each year. This would cost the city, if it were responsible for the whole expense of vaccination, at the rate now paid in Philadelphia for public vaccination, about $40,000 yearly—really a very insignificant sum when the enormous benefit to be derived is considered, but immense, as I have said, in the estimation of our legislators. Of course, in reality, a very large number of persons are vaccinated or re-vaccinated at their own expense; how many, there is no means of accurately estimating, but I should think that the city ought to be prepared to vaccinate or re-vaccinate at least 40,000 persons yearly, for although nothing like that proportion of persons accept gratuitous medical relief, a very large number who are willing to pay a physician when they are sick, will not incur the expense of vaccination to prevent a sickness which they hope may never come upon them. In fact, there were performed in Philadelphia during the ten years preceding 1880, in round numbers, 130,000 successful operations;[1] while, if my estimates are plausible, there should have been about 400,000, or as many as 270,000 more. Adding to the last number the average yearly estimate of 85,000 to be vaccinated, Philadelphia found herself at the beginning of 1880 with about 350,000 persons to vaccinate, in order to be thoroughly protected from smallpox. These figures are not presumed to be more than approximately correct, yet they are, perhaps, close

[1] Records of Health Office.

enough to the truth to show what a wide field in the direction of vaccination lay before the Health Board when confronted by the epidemic. The number of operations really paid for by the Board in 1880 (and for such only is compensation given) was but 28,415, so that it was hardly to be expected that the end of the year would witness the abolition of the epidemic. The unprotected persons were, however, scattered throughout the whole community, and this absence of aggregation is perhaps the reason why the devastation from the disease was not greater. Those persons especially received the care of the vaccine physician who, from contiguity to the sick, were most likely to be attacked by variola. Incomplete as I have shown were the means adopted to eradicate the disease, yet, in a letter which I wrote as correspondent to the "Bulletin" of the National Board of Health, I claimed a certain measure of success for them in instancing in comparison the course of the disease in the neighboring city of Camden, where even so much was not done. Smallpox was imported into Camden from Philadelphia in February, 1880, and caused there, as I was informed by Dr. James Wroth of that city, in the course of one year 131 deaths among a population of 41,638 inhabitants; the same proportion of deaths in Philadelphia would have given 2665 for that city instead of the 424 deaths recorded. The fact, however, is not to be lost sight of that epidemics are always liable to be more severe in small than large cities, probably from other causes than the want of sanitary protection.

The comparatively large number of deaths from *measles*, 108, the largest in any year since 1874, rep-

resents a great multitude of cases of the disease, since the affection is so rarely fatal, even with its complications. It is so contagious a malady that when it appears in a community, it runs among the children like wildfire; given its occurrence in a family of the poorer classes, who use much in common, it will not only attack, with very rare exceptions, all the children under the same roof, but spread to the next house, and so on, till in a very short time it has searched out every child in the neighborhood not protected by a previous attack of the disease.

Deaths from measles were reported from each one of the city wards but four (the Sixth, Ninth, Twenty-first, and Twenty-seventh), so that the zymotic was very widely diffused, as follows naturally from its very contagious nature. The aggregate of deaths caused by it were, however, comparatively so few, that when divided among the twenty-seven invaded wards, the numbers for each are too insignificant to admit of profitable comparison of the respective mortality of these small divisions of the municipality. If, however, groups of wards be taken together as single districts, those, for instance, defined above in discussing the distribution of the deaths from smallpox, it will be seen that the better-placed, "old city proper," had no fewer deaths than those other parts of the city which were the principal sufferers as regards smallpox. The mortality from measles was 5 in January, 20 in February, 38 in March, 29 in April, 9 in May, and 4 in June, when the epidemic may be said to have come to an end, though there were 3 deaths subsequently, namely, 1 in October and 2 in December. It will be noted that the deaths had a

small beginning in January, growing more numerous quite rapidly till the maximum was reached in March, the month distinguished beyond all others in this climate for fatal affections of the air-passages, and that they reached zero in the summer, the season when the same diseases have their lowest death-rate. Evidently those were the complications which heightened the mortality in this otherwise trivial disease.

There were fewer deaths from *scarlatina* in 1880 than in any year since 1872; indeed, including the latter year, there have been only four years since 1861 (at which time our records begin to be reliable), in which a lower death-rate from this fever was observed. Nevertheless, in the rural wards, to the north of the central city, the Twenty-first (Manayunk), Twenty-second (Germantown), and Twenty-third (Frankford and Bridesburg), the mortality from the disease was greater than during the previous year; in the last ward the deaths increased from 6 in 1879 to as many as 26, the latter giving a rate of about 10 deaths to every 10,000 inhabitants living. Tracing briefly in my report for 1879 the cause of scarlatina in previous years, I forecast this diminution of mortality in 1880, but also deemed an increase in the number of the deaths from scarlatina probable at the close of that year.[1] We shall see, by referring to the monthly mortality, that the latter actually took place (Table E): January 17 deaths, February 28, March 11, April 17, May 32, June 39, July 11, August 20, September 17, October 16, November 32, and December 50.

[1] Report of Meteorology and Epidemics for 1879. Transactions Coll. Phys. Phila., 3d Series, Vol. V., pp. 92–93.

We find that *diphtheria* caused 323 deaths in 1880, just 2 more than the number reported for 1879; its distribution through the city was not much different from that noted in the latter year, though the outlying Twenty-second and Twenty-third wards each counted more deaths from the zymotic; in the former (Germantown) there were 33 deaths where there had been 17 in the previous year, giving a death-rate of about 10 to the 10,000 inhabitants in 1880, and in the latter ward 20 deaths instead of 10, or a death-rate increased to 7.5 to the 10,000 inhabitants; but these figures are considerably above those which express the mortality from diphtheria throughout the whole city, which I have calculated to be 3.4 deaths to the unit of 10,000 inhabitants. The seasonal distribution of the disease was the usual one, more deaths in the cold, less in the warm weather; 97 deaths in the first quarter of the year, 72 in the second, 41 in the third, and 101 in the last quarter. The approach to equality in the number of deaths in the years 1880 and 1879 is characteristic of the rather sluggish course of diphtheria in Philadelphia, as it changes for better or for worse; this much dreaded disease has never taken the severe hold upon this community to which the records of some other cities bear witness.

As with diphtheria, the 303 deaths from *croup*, set down for 1880, approximate quite closely in number to the 291 from the same cause in the previous year. These deaths were scattered over the whole city, but the wards forming a group in the northeastern section of the city (Sixteenth, Seventeenth, Eighteenth, Nineteenth, Twentieth, and Thirty-first), and two wards in the southern part of the city (Second and Twenty-

sixth) were the greatest sufferers, the mortality in these districts being from 5 to 8 to each 10,000 inhabitants, while in the remaining wards of the city the death-rate did not exceed from 1 to 4 deaths in an equal number of people. Of the rural wards, the Twenty-second (Germantown) and Twenty-third (Frankford and Bridesburg) had only from 1 to 2 deaths in each 10,000 inhabitants, but the Twenty-first (Manayunk) had as many as 4 deaths in that number. There is nothing unusual in this distribution of the disease; the central sections of the city are wont to be less severely attacked by croup and some other of the zymotic diseases than the north-eastern and southern districts, while the outlying wards vary as regards its prevalence, being sometimes classed with the more unfortunate districts, and again being in company with the less afflicted central wards of the city.

The deaths from croup were divided among the quarters of the year in a proportion strikingly similar to that recorded for diphtheria, there being in the first quarter 107 deaths, in the second 62, in the third 42, and in the fourth quarter 91. The two diseases are so similar, if they be not identical, that the same case would be returned by one physician as diphtheria, and by another as croup; so that if they are really different diseases, their true relative proportion is hopelessly confused in the records.

The mortality from *typhoid fever*, which had been decreasing annually since 1876, in which year it will be remembered the deaths were double the yearly average, now in 1880 advanced to 495, an increase over the mortality of the previous year of 151 deaths,

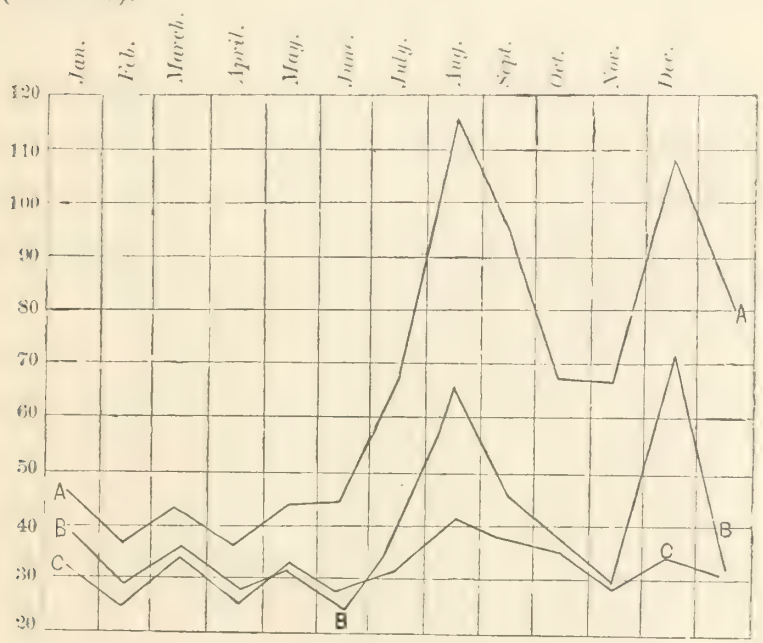
or 41 per cent. The death-rate, 5.8 deaths to each 10,000 inhabitants living, is, however, but very little more than the average mortality from typhoid fever during the ten years preceding 1876.[1] This increase in the mortality from typhoid fever in 1880 extended over nearly the whole city, since there were only five wards, the Eighth, Ninth, Tenth, Twelfth, and Twenty-sixth, in which there were fewer deaths from the fever than in the preceding year; and only two, the Eleventh and Twenty-third, in which the mortality was the same in the two years. The central wards, comprising the old "city proper" (Fifth, Sixth, Seventh, Eighth, Ninth, Tenth wards), were, generally speaking, the least severely visited by the disease; here, in a population of 112,848 souls, there were 44 deaths, or a ratio of 3.9 to the 10,000 inhabitants, while in the territory to the northeast (Eleventh, Twelfth, Thirteenth, Fourteenth, Sixteenth, Seventeenth, Eighteenth, Nineteenth, Thirty-first and Twenty-fifth wards), including the old districts of Kensington and Richmond, there were, in a population of 290,733, as many as 202 deaths, or a ratio of 6.9. In the southern section of the city (First, Second, Third, Fourth, Twenty-sixth, and Thirtieth wards), the ratio was but a little less favorable than the latter, being 5.9 deaths to each 10,000 inhabitants among 172,945 people. West Philadelphia (Twenty-fourth and Twenty-seventh wards) was nearly as bad as this, having a ratio of 5.2 deaths to the 10,000 with its 69,341 inhabitants. The Fifteenth, Twenty-ninth, and Twenty-eighth wards in the northwest had

[1] Rep. on Met. and Epidem., Trans. Coll. Phys. Phila., 3d Series, Vol. IV., p. 65.

death-rates from the fever respectively of 4.8, 6.1, and 3.5 to the same unit, while the outlying rural wards, Twenty-first (Manayunk), and Twenty-second (Germantown) had each rates of 6.6, and the Twenty-third (Frankford and Bridesburg) 6; the last ward has had this comparatively large share of mortality from typhoid fever for several years past.

The apportionment of the deaths from typhoid fever throughout the year followed the distribution which I have so often pointed out for them. The mortality in the several months was as follows: January 39, February 29, March 35, April 27, May 31, June 24, July 40,

Curves representing the monthly mortality of typhoid fever during 1876 and 1880, and the average monthly mortality of the same for ten years (1866–1875).



A. Mortality curve for 1876. *B.* Curve for 1880. *C.* Curve of averages.

August 65, September 46, October 38, November 29, December 72. A maximum occurred in August, and another in December, the latter appearing to have the most value, as happened really the previous year. I say appearing, because if we consult Table D we will see that the month of December has been made to embrace five weeks, while that of August contains but four, so that the excess in the former month is not a real one. I have repeated a diagram, which I drew for my report for 1876, containing a curve representing the monthly variations in the number of deaths from typhoid fever in that year, with another depicting the average monthly variations for the previous ten years, and I have added to them a third line to show the mortality from the fever in the several months of 1880. A truly remarkable similarity appears between all the curves, bearing witness to the constancy of "the periodical distribution" of the zymotic, and suggesting that this should always be taken into consideration when estimating the efficacy of any particular measures which may have seemed in some special instances to have successfully prevailed against the disease. The mild winter and the early and prolonged summer doubtless played an important part in bringing about the increase in the number of deaths from typhoid fever during the latter half of the year, as I attempted to show in my report for 1876 was the case under similar climatic conditions in that year.

There were reported a few deaths from *typhus fever* in Philadelphia in 1880, according to the death-list, 23; and that some of these, at least, followed true typhus is vouched for by physicians well qualified to

decide. The deaths were scattered, however, through several months (Table D), and were not numerous enough to excite apprehension of a serious epidemic.

The preponderance in the number of deaths from constitutional diseases in 1880, already alluded to, is in most part due to a greater mortality from tubercular diseases, and especially from *phthisis pulmonalis*. The deaths from tabes mesenterica bear a marked relation to climatic heat, but the connection of consumption with temperature is not so apparent; if in Table D the fact be kept in view that some of the months are made to include five weeks, while the others contain only four, the mortality from the latter disease will be found to have been divided not very unevenly through the year. Yet a certain connection will be observed between cold and an increased death-rate among the several months, but then there will appear a paradox in the fact, that in a year so exceptionally genial as we have seen 1880 to have been, the mortality from consumption exceeded that of the previous and some other years (Table C). The explanation, of course, is that there are other factors in determining this rate more potent than degrees of temperature; a truism, by the way, which should lead us to the practical inference that too much is not to be expected from a change of climate as regards the effects of the climate, *per se*, towards the cure of a case of consumption. Experience shows that though from year to year we may have such a variation in the mortality from phthisis as has occurred between 1880 and the previous year, yet there are not those wide differences that are found in the death-rates of zymotic diseases. Hence, even if it should be proved true that fell con-

sumption is allied to these in having for its cause some microscopic germ, it would seem that this poison is more constant in its degrees of virulence than those which occasion periodical epidemics of other diseases. We might reasonably explain the difference in the annual death-rates, since excesses of climate fail us, by changes in the modes of life—business pursuits, mental strain. It would appear then not unlikely that the revival of business, which began in the autumn of 1879, by creating new anxieties and giving incentive to overwork, was responsible for some additional deaths from phthisis pulmonalis.

The difference in the mortality from local diseases, the 7191 deaths in this class exceeding the number in the previous year by 769, seems to have been due in great measure to the disturbing influence of the early accession of summer heat. It belongs chiefly to the diseases of the nervous system, and, among these, especially to *cephalitis* and *convulsions*, terms often used by those who write death-certificates to express the causes of death in infants during the hot weather. The mortality from *sunstroke*, in the same class, is seen to be increased from 3 in the previous year to 37 in 1880, and of these as many as 23 to have occurred before the month of July (Table D), a very exceptionally early period for the climax of this disorder.

I presume, as with phthisis, the renewed activity in business will account for the larger number of deaths in 1880 classed under violent deaths; the risk of accidents is greater in the turmoil of busy life.

A CASE

OF

FUNGOSITIES OF THE BLADDER,

CURED BY SCRAPING WITH THE FINGER; WITH SOME REFERENCES TO THE LITERATURE OF THIS AFFECTION.

By

WALTER F. ATLEE, A.M., M.D.

[Read March 1, 1882.]

This case is reported because it is a rare one, because it is instructive in a practical point of view, and because consultation with most of his works of reference would not assist the surgeon in benefiting a similar case as much as can be done by doing as was done here.

Miss S. B. consulted me in the summer of 1880, on account of painful and frequent micturition, with hæmaturia. She was born in April, 1861; her father is a large, strong, and healthy man; her mother died when she was a child, after having suffered from many manifestations of scrofula.

She said she remembered to have felt, occasionally, a slight pain in passing urine, from her earliest recollection. When seventeen years of age she first suffered severely; her urine then was very light in color, with no sediment, but with a strong odor. The pain was while passing the urine and after it had passed; after the emptying of the bladder there was a constant desire to pass something more. She became thin,

pale, and haggard. When eighteen years of age, some pus appeared in the urine, and occasionally a little blood. These symptoms increased to such an extent that she was obliged to keep her bed for several months. This rest, aided, perhaps, by medical treatment, for she had always enjoyed the care and attention of our most experienced physicians and nurses, made some improvement in her condition, so that she was able again to go out. All the worst symptoms, however, soon returned, and when I saw her, in September, 1880, her state was a very serious one; there was constant inclination to empty the bladder, which could not be resisted oftentimes for more than a half-hour, and the loss of blood was considerable.

The urine, when examined at this time, showed pyoid bodies, epithelium from the bladder, and crystals of triple phosphate, together with blood-corpuscles in abundance. The blood was of a bright-red color, showing that the urine had not had time to produce those changes in color always produced by long contact with the hæmoglobin of the corpuscles.

With such symptoms, this case seemed clearly to be one of foreign body in the bladder, and the advice given was, to attempt its removal without delay. For this purpose the patient was put under the influence of anæsthetics, and the urethra was dilated by means of a pair of ordinary dressing forceps, introduced, opened, and withdrawn as often as necessary—this being, in my experience, the best way of effecting this dilatation.

When the finger was then passed into the bladder, nothing abnormal was felt, no calculus nor distinct tumors, except that about the fundus were a number of fungosities or soft growths, some of them more than a half inch in length, and about one line in thickness. These were carefully scraped off by the end of the finger and by the finger-nail. This simple operation resulted in the perfect cure of my patient, and until the present time there has been no symptom of a return of her disease.

I call the growths thus removed fungosities, and not villous growths, inasmuch as they were not like tufts of fine hair (*villi*), but resembled rather *fungi*, or certain mosses. I have several times seen similar growths removed from the cavity of the uterus, in cases where for years they had been the cause of alarming hæmorrhage. They have, anatomically, the same fundamental structure as the mucous membrane whence they spring; they are simple excrescences of this membrane; they are formed of granular amorphous matter, of cellular tissue in small quantity, and of fibro-plastic elements; almost all have a large number of capillary vessels ramifying through them, and some are covered by epithelium. This epithelium is *on* the surface, *homologous*, and not *within* the subjacent connective tissue, *heterologous*, which is characteristic of epitheliomatous growths.

I said that one reason for reporting this case to the College was, that consultation with most of his works of reference would not enable the surgeon to benefit his patient as much as was done in this case. In Holmes's *System of Surgery*, it is said: "The indications are to allay pain, to subdue spasmodic action of the bladder, to prevent hæmorrhage by internal remedies, and to counteract its effects on the system by chalybeates and nutritious diet. Astringent injections very carefully introduced into the bladder, such as weak solutions of acetate of lead, or of nitrate of silver, may be tried; they are, however, not to be repeated more than once or twice, unless marked benefit is observed, and signs of vesical irritation have not been produced by their employment." This is the advice given in almost all surgical works, not only

general but special. Even in Coulson's excellent work on *Diseases of the Bladder and Prostate Gland*, of which a sixth edition was published in 1881, we find nothing but a similar plan of treatment recommended. Though quite out of place, I will cite here the diagnostic symptoms given in Coulson's work between villous growths and calculus, as being the best and clearest I have ever met with. In all works the diagnosis of these growths is said to be extremely difficult; for example, in the *Dictionnaire de Médecine*, it is said, "Fungus of the bladder may be suspected, but a precise diagnosis cannot be established"—tome xxx. p. 744. "The pain in calculus is most severe after the urine has been passed, but in villous tumors the discomfort is aggravated by fulness of the bladder, and relieved by its evacuation. The pain in calculus is relieved by rest, which has little or no effect upon the symptoms of tumors of the bladder. The hæmorrhage also in the latter affection is neither decidedly aggravated by movement nor relieved by rest. In villous growths, the blood is generally pure; in hæmaturia, due to calculus, there is generally more or less pus mixed with the blood. Examination by the rectum, or with a sound in the bladder, causes pain in cases of villous growth, and increases the hæmaturia, whereas the symptoms of calculus are not necessarily aggravated by these manipulations."

To return to treatment, Nélaton says: "In women, it is sometimes possible to reach the fungus by dilating the urethra and the neck of the bladder. The case of Warner tying a polypus of the bladder in this way is recorded above; and in case of a fungous

growth, an analogous operation should be done"—*Pathologie Chirurgicale*, tome v. p. 301.

In Warner's case, above referred to, a polypus penetrated into the urethra of a young woman, and pushed out of the meatus. An incision was made, dividing the half of the urethra, the rest was dilated, and also the neck of the bladder, the tumor was drawn out, and a ligature applied to the pedicle.

In the *Principles and Practice of Surgery* of Professor Agnew, and in the third edition of Professor Gross's treatise on *Diseases of the Urinary Bladder*, revised by Dr. Samuel W. Gross, we are advised when symptoms of papillary and polypoid fibromas are seen in women, to dilate the urethra by special instruments, and remove them as may be found best under the circumstances of the case. Some eighteen cases, altogether, are recorded in these works where vesical growths were removed by various operations. Of the prognosis in such cases, Dr. Gross forcibly and truly says: "It is of the worst possible description. Death almost invariably follows from sheer loss of blood, or the combined effects of hæmorrhage and pain." Of the treatment, he says, "surgical interference is demanded imperatively, since, without it, a fatal issue is almost the inevitable result."

The best account I have met with of the flocculent excrescences or fungosities in the bladder is contained in the *Lectures on the Surgical Disorders of the Urinary Organs*, by Reginald Harrison, 2d edition, London and Liverpool, 1880. Mr. Harrison refers to the paper of Robert S. Hudson, in the *Dublin Journal of the Medical Sciences* for June, 1879, to that of Professor G. Murray Humphrey in the *Medico-Chirurgi-*

cal Transactions for 1879, which contains probably the best account to be found of the pathology of growths into the bladder, to Mr. Norton's cases in vol. xii. of the *Clinical Society's Transactions*, and to a paper of J. H. Roberts and C. De Morgan in vol. xxi. of the *Transactions of the Pathological Society*, where the microscopical appearances are very beautifully represented. He also relates a case (p. 359) communicated to him by a Dr. Alexander, where chloroform was given and the urethra dilated; a wire écraseur was passed, and a large growth was removed; smaller growths were removed by the finger. Fifteen months afterwards it was necessary to remove some more by the finger, after which the patient remained well.

This case of Dr. Alexander is doubtless the same as that related in great detail in the *London Lancet* for August 17, 1878. The writer says he cannot find any other case recorded of removal of a villous growth from the female bladder, and quotes from *Bryant's Surgery* "that there is no cure for this affection; the surgeon can only relieve symptoms. The disease usually destroys life in about two years."

Enough has been said to show that cases such as I here report are rare; that they cause great suffering, and, eventually, loss of life; that the means usually recommended fail in giving relief; and that an operation, easily performed, attended by no risk, and followed by no bad consequences, does cure such cases, and that this operation appears to be very generally unknown.

One more observation may be permitted, indeed seems called for, in this rather desultory paper. This

is, that the history of a case such as is here related, justifies us in looking favorably upon the resort to a similar proceeding in cases of similar disease in the male patient. An incision into the neck of the bladder, when so much suffering and so great danger to life are present, is surely justifiable. It is a matter of no great difficulty or danger. Even if it was found, after the making of this opening, that the diseased tissues could not be taken away, the patient would, in all likelihood, obtain some relief, from the free passage afforded to the purulent and bloody discharges. There is a case recorded (see *British Medical Journal*, vol. ii., 1875, p. 493), where Billroth did this, and encountering a fibrous tumor, the size of which was such that it could not be extracted through the perineum, he cut through the recti muscles above the pubic bone, made a transverse incision into the bladder, and then tore through the tumor near its base with the finger, and dissected out the pedicle; the patient was perfectly cured.

TWO CASES

OF

CONGENITAL IRIDEREMIA, WITH LAMELLAR CATARACT IN ONE AND DISLOCATED CATARACTOUS LENSES IN THE OTHER.

By

GEO. C. HARLAN, M.D.,

SURGEON TO WILLS EYE HOSPITAL AND TO THE EYE AND EAR DEPARTMENT OF THE PENNSYLVANIA HOSPITAL.

[Read April 5, 1882.]

Case I. N. J. M., aged 12, says that he knows of no defect in the eyes of any other member of his family in this or preceding generations, except that his mother is "short-sighted." He is of medium size and, except his eyes, well developed.

His vision is $\frac{5}{cc}$ and is not improved by glasses or the stenopæic hole. There is no photophobia, and he sees better in a bright light than in a subdued one. The corneæ are clear, but perhaps very slightly below the normal size, though a constant and decided nystagmus makes it impossible to measure them accurately. The eyes are free from irritation, and their tension is normal. There is not a vestige of iris in either eye. There is well-marked lamellar cataract in both eyes. In each lens there are two opaque layers with clear lens matter intervening between them, beautifully seen by oblique illumination. Only occasional and partial glimpses of the fundus can be obtained, but the choroid seems nor-

mal. There is not sufficient vision for any satisfactory test of the accommodation.

Case II. I. B., a well-grown boy of 13 years, has rather prominent eyes, with full-sized corneæ, and normal tension, but the iris is entirely absent in both. In the right eye the cornea is quite clear, and, though there are floating opacities in the vitreous, the details of the fundus can be seen fairly well. The lens, which is of normal size, is opaque and white, and is dislocated upwards, so that only about its lower half is seen beneath the cornco-sclerotic junction. The ophthalmoscope shows a hypermetropia of $\frac{1}{6}$, which, assuming $+\frac{1}{4}$ to represent the loss of refractive power induced by aphakia in the emmetropic eye, indicates a structure of the ball corresponding to a myopia of $\frac{1}{12}$. The choroid and retina appear normal in structure, but the optic disk is atrophied and greatly distorted, the vertical diameter being twice that of the horizontal, and the retinal vessels crowded to its inner edge; vision is only quantitative. There is no nystagmus.

In the left eye there is a diffused haziness of the cornea, which, together with a greater opacity of the vitreous than in the other eye, makes the details of the fundus invisible. The lens, which presents the same form of cataract as that in the right eye, is still further out of position, and only a narrow edge of its periphery extends below the margin of the sclerotic. The lenses seem to be held in their unnatural position by some attachment of their upper margins, while the lower are free, and they swing backwards and forwards, as if on hinges, with the movements of the balls. In the left eye the lens has caused an absorption of the tissues, against which it rests until the choroid has disappeared from above it and the sclerotic is very much thinned and slightly staphylomatous. When the cone of light concentrated by a convex glass is thrown upon it from below, the outlines of the lens become distinctly visible through the sclerotic.

Though irideremia (iris and eremia, absence), or aniridia, is an extremely interesting anatomical curiosity, it can scarcely be said, in the present state of our knowledge, to teach any useful lesson in embryology or pathology. Numerous hypotheses have been suggested to account for its occurrence, but most of them are more fanciful than philosophical, and perhaps none of them are more rational than that maintained long ago by Von Ammon, who, in view of the late appearance of the iris, after the choroid is fully formed, considered its absence as simply the result of an arrest of development of the uveal tract. It is a curious fact that this anomaly is almost invariably symmetrical. According to Manz (Handbuch, Augenheilkunde II. p. 90), but one case has been reported in which it occurred in one eye only. Another point of interest in the history of this defect is its decided tendency to hereditary transmission. A number of cases of inheritance have been reported; one (quoted from Von Ammon by Lawrence) in which one member of the first generation was affected, three of the second, and five of the third.

The fact that cataract is very generally associated with irideremia, has given some support to a suggestion that the iris is concerned in the nourishment of the lens: and to an ingenious theory of the causation of irideremia which supposes that the iris has been crowded out, as it were, by the lens remaining too long and too closely in contact with the cornea during embryonic life. This coincidence of cataract with irideremia is, however, not constant, as cases are recorded in which the lens was transparent. One is

reported by Dr. Reuling (*Am. Journ. Med. Sci.*, Jan. 1875), in which vision was sufficiently acute to enable the observer to determine that the power of accommodation was unimpaired.

The normal intraocular tension found in cases of irideremia has been adduced to prove that the aqueous humor is not secreted by the iris, but chiefly, if not entirely, by the ciliary processes. In the rabbit the ciliary processes are connected with the iris, and when both are removed, the eye becomes very soft and the aqueous humor is never regenerated (Deutschman, *Graefe Arch.*, v. 26, No. 3). This, so far as it goes, gives support to the advocates of sclerotomy, instead of iridectomy, in glaucoma.

Partial congenital luxations of the lens are usually upwards, as in Case II., and frequently occur without defect of the iris. In the left eye of this patient, the lens is so far out of place that it would be entirely concealed if the iris were present, and this case might then be readily mistaken for one of the very rare anomalies of congenital aphakia.

[After the reading of the preceding paper, Dr. W. F. Norris said:—]

Through the kindness of Dr. Harlan, I have had an opportunity of making an examination of the eyes of the patients whose cases have been described. On first inspection of one of the cases I thought I could detect a slight peripheral remnant of the iris, but more careful examination, aided by the ophthalmoscope and oblique light, showed that every vestige of iris was wanting, and that this appearance was due to the shadow cast by the limbus conjunctivæ corneæ on the periphery of the anterior chamber.

It has occurred to me that at least some of the cases which have been reported in the older books as partial irideremia, might possibly be due to this similar appearance in the days when the above-mentioned methods of examination were unknown.

REPORT

OF

A CASE OF MALARIA, IN A CHILD AGED 20 MONTHS, IN WHICH MORBID ENLARGEMENT OF LIVER AND SPLEEN OCCURRED, AND ALSO OF A CASE OF AORTIC STENOSIS AND REGURGITATION WITH ATHEROMATOUS AORTA IN A WOMAN AGED 103.

WITH EXHIBITION OF THE SPECIMENS.

By

JOHN M. KEATING, M.D.,

LECTURER ON DISEASES OF CHILDREN IN THE UNIVERSITY OF PENNA.

[Read April 5, 1882.]

The following notes were taken for me by Dr. Charles E. Bricker, Resident Physician at the Philadelphia Hospital. I give the statement of the mother, as throwing some light on the antecedent history of the child, from whom the interesting pathological specimens were taken which I have the honor of bringing before the College to-night:—

Case I. M. N., aged 26, married, entered the obstetrical wards of the almshouse two years ago, shortly after her arrival from Ireland. Little more than a month after, July 24, the child was born. She remained in the house one year, and went to Wilmington, Del., to live. Shortly after her arrival there, in July of last year, she was seized with chills;

she states that she lived in a swampy locality, and that everybody living in the neighborhood suffered severely from intermittent fever. She states that the malarial paroxysms were not of the usual type, the disease being often of a cerebral form, accompanied by violent headaches, which compelled her to place ice to the head, and that she was at times delirious. The attacks came on daily for several days at a time, commencing at four o'clock in the afternoon. She nursed the child until its death. About two weeks after the mother was first affected the child began to fail—it exhibited the same symptoms as the mother. Its attacks would come on about four in the afternoon. The ears became cold, finger nails blue, there would be vomiting, followed by frequently loose stools of a green color, and containing a great deal of mucus; at times constipation would exist for days, to be followed by stools of a chalky color. There were no convulsions; no strabismus. Child always nursed well, and the mother always had plenty of milk. On the 15th of October following, the child being fifteen months old, she again sought admission to the hospital. Upon admission the mother showed a marked malarial cachexia, which was also equally noticeable in her infant. The mother was placed upon iron and quinia in large doses, followed by cod-liver oil, upon which she rapidly improved. Her milk was examined microscopically, and found to be normal. On examination of the child, attention was immediately attracted to the abdomen, which seemed to be unusually enlarged, especially upon the left side; this was found to be due to an enlarged spleen, which extended from the normal area of splenic dulness down to the crest of the ilium, its anterior edge terminating in a ridge, about an inch to the left of the umbilicus. The mother had not noticed this ridge until her admittance to the house. The liver was also found to be enlarged, and the intestines were unduly distended with gas. Every effort was made by treatment, both constitutional and local, to reduce the liver and spleen to their normal size, and for a time we thought our efforts would be successful, as the lower border of the

spleen became separated from the ilium by a line of tympanitic resonance. The malarial attacks were checked by suppositories of quinia, but the use of tonics and judicious diet had little effect upon its nutrition. The child died March 5, 1882, aged twenty months. Post-mortem examination gave us specimens, which I bring before you—liver weighed 19 ozs., and the spleen 14 ozs.

There was no history of syphilis in this case. Dr. Formad expressed the opinion that the enlargement of the spleen was that of simple hypertrophy of the congested organ, due to malaria; the enlargement of the liver being due to fatty change; the specimens, on this account, are of great interest.

The following notes were given me by Dr. W. A. Edwards, Resident Physician at the Philadelphia Hospital:—

Case II. Mary Anderson, aged 103, was admitted to the old colored woman's ward January, 1882. About three weeks after admission she suffered from an attack of bronchitis, which persisted more or less until her death; while treating her for this affection, attention was called to her heart, which was found to present a well-marked, double aortic murmur, *i. e.*, regurgitant and obstructive, also a slight mitral whiff, probably regurgitant. She presented no lesion of any other organ, complaining only of slight rheumatic muscular pains. The patient was well nourished, appetite good, cerebral condition fair. She continued in the above condition, gradually failing, until death, which occurred March 22, 1882.

Post Mortem.—Lungs: Bronchitis well marked over both lungs; the bronchial tubes and bronchioles exuding pus, upon pressure, in noticeable amount. At the right apex were a few masses, which had undergone calcareous degeneration, cicatrization, and puckering. The point at which

they were deposited was drawn into a stellate mass attached to the pleura. *Heart* and *Aorta:* Upon removing these organs they were found to present a very interesting condition of atheroma, due to the age of the patient, and the unusual duration of the disease, which enables us to study it in what may be termed its last stages. The aortic valves showed atheromatous changes, the middle leaflet being almost solid at its base; the aorta was dilated, and thickened with atheromatous deposit almost concentrically surrounding the vessel; portions of this, when detached, were like thin sections of solid slate. Along its course, six inches from the aortic valve, there was a large patch of ulceration, on which blood had collected into a firm organized clot, thus preventing rupture. The coronary arteries may be felt like twigs of wood in the heart muscle.

The interest of this case lies in the fact that the supposed great age of this patient was arrived at, by the absence of laborious occupation—and the fact that with the nature of her heart lesion, sufficient compensation had been established to carry on life, no extra strain having been placed upon the heart's power. This case is an interesting study in prognosis.

OBSERVATIONS ON CATARRHAL FEVER.

By

J. M. DA COSTA, M.D.,

PROFESSOR OF PRACTICE OF MEDICINE IN JEFFERSON MEDICAL COLLEGE.

[Read May 3, 1882.]

Those engaged in active medical practice must have been struck with the widespread prevalence of an epidemic of catarrhal fever presenting many curious features. As it is only by a study of all such epidemics that we shall ever learn fully to understand this dissimilar malady, I trust it may not prove without value to record before the College my individual experience with it.

The disorder begins almost invariably in a sudden manner, sometimes with a chill, quite as often without it. I have known persons well in the afternoon, and in the evening with a decided fever, and suffering all the discomforts of the catarrhal malady. Among the first signs of this are pain in the throat, and a feeling as if it were filled up, yet looking at it nothing is seen but redness and some relaxation. Fever is by this time developed, at first of only moderate intensity and with a quick but very compressible pulse. Dry cough soon becomes a feature, occurring not infrequently in paroxysms, and now and then combined with loss of voice, and with difficulty in swallowing. The chest walls are sore, and the cough is painful.

Frequent, rather difficult breathing, not associated with any marked physical signs except feebleness of respiratory murmur, is a common symptom. As the malady progresses, more obvious signs of bronchial catarrh may happen, and harsh breathing and dry rales be found on listening to the chest. But here and there will be a spot still marked by feeble breathing, a spot of seeming congestion of the lung and of impaired expansion. Scanty tenacious sputum, blood streaked, is perhaps noticed, to become more copious and purulent only in cases in which the bronchial catarrh is prominent. The eyes are, as a rule, injected or watery, but nasal catarrh does not exist. Yet late in the disease it may come on, and the malady pass off, in the language of the patient, by a bad cold in the head. Besides these catarrhal symptoms, are pains,—chest pains, pains in the neck and scalp, pains in the loins and limbs. The chest pains are most peculiar and severe. They are sharp and like pleurisy, indeed they are so regarded. But only impaired respiration exists, friction does not, save in the rarest instances; and the character of the pain, its having its seat in the chest walls, is shown by its transferring itself with rapidity from one side to the other.

The state of the skin is at first dry and harsh. It becomes soft and clammy as the disorder advances, and copious sweats, especially at night, are common. The face at the outset is apt to be flushed, and what has particularly struck me in this epidemic as a feature which I cannot recall to have noticed so strikingly before, is a curious irregular mottling of the surface. This is very marked on the neck and breast, and might easily cause the case to be mistaken for

scarlet fever or for German measles. But when closely looked at, it is seen how the capillary injection is really quite unlike the eruption of either.

As temperature observations on catarrhal fever are very imperfect, I recorded whenever a good opportunity offered as many as possible. Here is a case in which with the aid of a very intelligent nurse they were made three or four times daily, and begun a few hours after the first symptoms had manifested themselves.

1*st day.* 2.30 A. M., 102.2°; 6.45 A. M., 100.8°; 8 P. M., 101°.

2*d day.* 11 A. M., 100.8°; 9 P. M., 101.6°.

3*d day.* 2 A. M., 104°; 10.30 A. M., 101.5°; 3 P. M., 101.5°; 6 P. M., 103°; 9 P. M., 105°.

4*th day.* 7 A. M., 101.5°; 9 A. M., 99.5°; 11 A. M., 100°; 6 P. M., 100°.

5*th day.* 11 A. M., 99.2°; 9 P. M., 99.6°.

6*th day.* A. M., 99°.

7*th day.* A. M. 98.5°, no evening rise.

This temperature was the highest I have met with in an uncomplicated case. It attained its height on the third day, and is seen to be very irregular. In truth, irregularity of temperature is one of the characteristic features. The temperature is apt to be irregular until the whole disorder markedly declines, when it by gradual degrees, but in the space of a day or two, returns to the normal.

Next to the catarrhal and febrile symptoms the gastro-intestinal claim attention. Disgust for food, pasty tongue, are very usual, and attacks of diarrhœa not unusual. In some cases, indeed, the intestinal

catarrhal symptoms are far the most prominent, and it may be that only with their subsidence the bronchial catarrh appears. Nor is it always a simple diarrhœa. Seizures bespeaking an irritation of the large intestine, diarrhœas soon merging into dysenteries form quite a fair proportion of the cases. The urine is high colored, scanty, but free from albumen, even in cases with a temperature of 105°. Only in instances of most marked pulmonary congestion have I known it to contain albumen, and then but in small quantities.

As regards the nervous symptoms, great lassitude, restless nights, and marked hebetude strike us most. With reference to the drowsiness, it is often so decided, that it is difficult to believe that the patient has not taken opium. Delirium I did not once encounter; nor were the cutaneous hyperæsthesiæ as common as I have noticed them in other epidemics. In truth, on the whole, the nervous phenomena, except the hebetude, were less pronounced.

The duration of the disease is a short one. It does not, unless kept up by complications, exceed a week; nor did I see a fatal case, unless from complications. During the rather tardy convalescence, what forces itself on our attention is the weakness with the decided loss of flesh which so short a disease has occasioned. Of course, I am speaking only of marked cases, and not of the slight ones of a few days' duration that abound as a light manifestation of the epidemic influence. Glandular enlargements are very occasionally met with during the convalescence; more often did I notice inflammation of the antrum with its distracting headache and sense of fulness and pain.

I have just alluded to the complications. Pneumonia, catarrhal and lobar, is the most common. And I am quite clear that the great prevalence just now of pneumonia must be mainly ascribed to the influence of the poison of the catarrhal fever. But this is too large a question to enter into here; as it would equally lead me too far to inquire whether there are any clinical differences which separate these pneumonias of epidemic origin from those originating from other causes.

Besides pneumonia, I have met with overwhelming attacks of pulmonary congestion. One, for instance, seen with a medical friend, in which a bright lad of sixteen perished who had not been ill forty-eight hours; perished with bloody tenacious sputum, temperature of 104.8°, intense dyspnœa, heavily congested lungs terminating in œdema, and amid vanishing pulse, wild struggles for life, and signs of non-aerated blood—in whom nevertheless there were no spots of dulness or bronchial breathing or other evidences of consolidation to be detected. Then I saw with Dr. Herbert Norris, in a previously healthy, although rather delicate, young woman, who was seized with catarrhal fever just as her little girl was fairly convalescent from it, rapid phthisis develop itself; primarily in the lung which three or four days after the acute setting in of the catarrhal malady had slowly advanced to imperfect consolidation at its lower part, then more rapidly in the right. On the side first affected a large cavity formed in the lower lobe and became manifest on about the twelfth day of the disease. The whole duration of the case was just

three weeks; the only instance of tubercular affection to be traced in the family was that of an aunt.

Another complication I have met with is gangrene of the lung. I saw such a case with Dr. Girvin. The sputum was horribly offensive, the wasting decided; a spot at the upper part of the left lung was gangrenous. These symptoms had set in acutely, about ten days after an attack of catarrhal fever in a young woman before in good health.

There is generally little difficulty in the diagnosis of the epidemic malady; the catarrhal symptoms, the signs of the general disorder, are very manifest. Occasionally a puzzling case happens; as for instance, one in a young girl with nose-bleed, with diarrhœa, with high temperature, all within the first week of the disease. Yet the sequence of the phenomena prevented the affection from being mistaken for typhoid fever. The nose-bleed came on after the marked catarrhal symptoms; the diarrhœa appeared on the fourth day, and lasted only forty-eight hours; the high temperature continued but for a day, and then there were very irregular variations until, by the eighth day, the temperature had declined to normal.

One of the most interesting features of the present epidemic is its infectious character. In one household five members took it in succession; in another, it began with grandchild and ended with grandmother, after two children, mother, and three servants had had it. Nor are those exempt who are confined to the house. One of the most marked cases I encountered was in a lady who has been for five years

bedridden; in another, the patient had not been out of doors for ten months.

As regards the treatment I have nothing to add to what is well known. It has to be symptomatic; and in the very young and the very old decidedly supportive. My experience, however, makes me urge the advantage of employing quinine almost from the start; and has taught me that small, repeated doses of opium have a most happy, steadying, and distress-allaying influence.

CASES

OF

POISONING FROM DRINKING IMPURE WATER.

By

JAMES H. HUTCHINSON, M.D.,

ONE OF THE ATTENDING PHYSICIANS TO THE PENNSYLVANIA HOSPITAL.

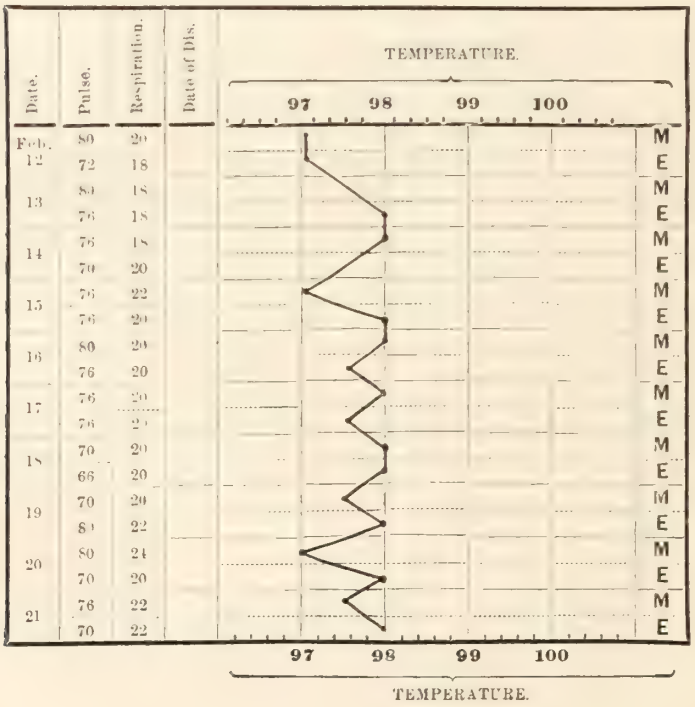
[Read May 3, 1882.]

During the past three months I have had under my care at the Pennsylvania Hospital a series of cases, which presented a rather unusual set of symptoms, and which possessed so much interest that I feel justified in taking up your time this evening with a brief account of them.

The patients were all Germans, and were all employed in a large sugar refinery in this city at the foot of Bainbridge Street—on the Delaware River front; where, however, none of them had worked more than two, or at most three weeks before the occurrence of the attack. The cases so closely resemble one another, that the history of one will suffice for my purpose. I have selected that of the first patient admitted, which was, moreover, much the severest of them all.

R. R., æt. 21, born in Germany; single; shoemaker, was admitted into the men's medical ward of the Pennsylvania Hospital on January 30, 1882. One of his sisters is

said to have died of consumption, but with this exception his family history is good. His own health has always been good until two weeks ago, when, without any apparent cause, he was seized with diarrhœa, attended with a good deal of prostration. He has had as many as ten to fourteen stools a day, and has also had at times nausea and vomiting, but is sure that he has never had fever. Yesterday the diarrhœa ceased, and he has had no passage since. Upon admission the patient is anæmic, and complains of excessive weakness, of vertigo, and of pains in his back and limbs. The tongue is dryish, and coated with a thin whitish fur; the appetite poor, and the pulse slow; the temperature subnormal, 97°; there is no tympany. The examination of the heart, lungs, and urine gives negative results only; he was ordered a pill containing nitrate of silver gr. $\frac{1}{3}$, and opium gr. $\frac{1}{4}$ three times daily, and placed upon a restricted diet.



Feb. 2. The patient's bowels have not been moved since his admission; he feels less weak than when admitted; his tongue is also less coated, but his temperature is still subnormal. The use of the pill was discontinued, and he was ordered a teaspoonful of Huxham's tincture three times daily.

Feb. 20. The bowels are now moved regularly; he does not appear to gain strength; he was, therefore, ordered cod-liver oil.

March 6. Discharged cured.

I confess that when this case first came under my care, I was puzzled how to explain the symptoms it presented. The presence of excessive prostration with a history of diarrhœa lasting for two weeks, seemed to point to typhoid fever as their most probable cause. Against this hypothesis was the fact that the patient was positive in his assertions that he had not had fever at any time since he was taken sick, that none was present at the time of his admission or subsequently, and that there was no tympany or rose-colored spots. His condition called, however, for a supporting treatment, and I thought it best to restrict his diet to liquid articles of food.

The other cases came in rapid succession, and presented the same symptoms as the first case. There were in all of them prostration, slow compressible and often dicrotic pulse, subnormal temperature, diarrhœa, sometimes accompanied by slight pain in the bowels, and absence of tympany. This similarity of symptoms appeared to indicate an identity of cause, and it was soon discovered that in addition to having been employed in the same refinery, they had been all subjected to an influence which seems to me sufficient to explain the symptoms from which they suffered.

It was found upon inquiry that water from the river Delaware is introduced into the refinery for use in cleansing the evaporating pans, and that, after being used for this purpose, it is returned to the river above the point although at a considerable distance from that at which it is taken.[1] The water is distributed throughout the building, and being more accessible to the workmen than the ordinary hydrant water is freely used by them for drinking purposes. The water, although said to be filtered before being put to the use first referred to, is probably from the presence of organic impurities unfit to drink.

There can be but little doubt that the use of the water of the Delaware River, contaminated as it must be opposite Bainbridge Street by sewage, was the cause of the singular symptoms observed in the six patients who came under my care. Impure as it was, it did not, however, produce typhoid fever, and my cases show that while impure water may produce prostration as great as that seen in this disease, it will not produce the fever itself, unless it contains the specific typhoid germs.

The symptoms presented by these cases seem to have indicated the existence of blood poisoning rather than of irritation of the bowels. The latter could hardly have existed without the presence of a certain amount of fever. The temperature records will show that at no time in any one of the cases was there any elevation of temperature above the normal. The stools, too, indicated the existence of relaxation rather than

[1] The water is taken from the foot of a long wharf projecting into the river; it is returned at the point nearest the factory. These two points are distant from each other about 150 feet.

of inflammation: they were rather dark in color and watery, and never presented the ochrey yellow appearance of the stools of typhoid fever. The cases further show that persons may become accustomed to the use of an impure water, or that at all events it may cease to excite in them after a certain time any active symptoms. It will be remembered that all the patients had only recently been employed in the factory, and the effects of drinking the water had shown themselves very soon after they had entered upon their duties. The older hands drank the water with impunity.

The cases all did well, and required little treatment. Hope's camphor mixture being used in most of the cases to check the diarrhœa, and quinia or Huxham's tincture as a tonic.

It may be well to add that the attention of the proprietors of the refinery has been called to the supposed cause of the illness of their workmen, and they have taken the proper precaution to prevent the occurrence of further sickness among them by cautioning them against the use of this water for drinking purposes.

I have appended to this paper the histories of the remaining five cases, which I had the opportunity of observing. They are drawn up from notes taken by Dr. J. M. Fox.

Case II.—Diarrhœa with typhoid prostration, A. W., æt. 18: born in Germany, single, baker; admitted March 1, 1882; discharged March 13, 1882, cured. He came to this country last September; has always been very healthy until two weeks ago, when he began feeling weak and miserable, and to have headaches and diarrhœa, which have continued.

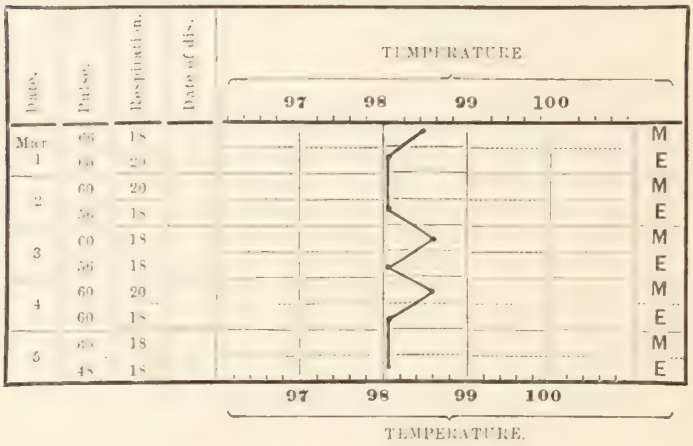
Upon admission has no fever; temperature subnormal;

appetite poor; tongue slightly coated; some hebetude and slight headache; no fulness of abdomen; a few râles heard over apex of right lung; examination of heart and urine negative. Ordered quinine gr. viij, and liquid diet.

2d. Tongue clean; only one passage from bowels; pulse slow, with a tendency to reduplication.

3d. Bowels not moved since; no fever; feels much better, but is still rather dull.

13th. Continued to improve, and is now well.



Case III.—Diarrhœa with typhoid prostration, H. S., æt. 28; born in Germany, single, laborer; admitted March 13, 1882; discharged March 20, 1882, cured. Has generally been very healthy; eight days ago he began to have diarrhœa, pain in the stomach and limbs, headache, and vomiting, and these symptoms have continued; has had as many as twelve loose stools a day.

He has been working in a sugar refinery, and is the third patient who has been admitted from there into the hospital within the last month and a half, with much the same symptoms, except that the prostration of the other two (R. R. and A. W.) was more marked.

He says that he has been in the habit of drinking the

water that is pumped from the Delaware, which is very likely the cause of the trouble.

Upon admission patient feels weak and dull, has headache, vomiting, and diarrhœa; pulse weak and reduplicated (this was also the case with the other two patients); appetite good; tongue clean. Examination of heart, lungs, and urine gives negative results. Hope's camphor mixt. f℥ss. was administered as needed.

15th. Bowels are still loose, and passages are light colored; no more vomiting; diet has been restricted; camphor mixt. was stopped, and powdered opium gr. ¼, with oxide of silver gr. ⅓ were given three times daily.

19th. Diarrhœa has been checked, and he now feels and looks quite well. His temperature since admission has ranged from 98° to 98½°.

Case IV.—Diarrhœa with typhoid prostration, F. S., æt. 34, born in Germany, laborer; admitted April 17, 1882; discharged April 25, 1882, cured. Has generally been very healthy. Two weeks ago, while working at a sugar refinery, he was taken with pain in the bowels and diarrhœa, which have continued up to the present time.

Upon admission patient was somewhat prostrated; has diarrhœa and tenderness in the epigastric region; the tongue is slightly coated in centre, moist; appetite poor; pulse slow; no fever. Heart, lungs, and urine normal. Ordered quinine gr. viij. daily. Hope's camph. mixt. f℥ss. as needed, and liquid diet.

April 20. Diarrhœa has been checked, and he feels much better.

25th. Feels very well; bowels regular; appetite good. Temperature has ranged from 98° to 98½°.

Case V.—Diarrhœa with typhoid prostration, M. Z., æt. 29, born in Germany, laborer; admitted April 17, 1882; discharged April 25, 1882, cured. Eight days ago, while working at a sugar refinery, he began having diarrhœa and pain

in the bowels, and these have continued up to the present time.

Upon admission his face is slightly flushed, and he has a typhoid appearance; is somewhat prostrated; tongue coated in centre, moist; pulse slow; bowels were opened six times during the night. He has no fever, and there is no fulness of the abdomen. Examination of heart, lungs, and urine gives negative results. Ordered Hope's camphor mixt. f℥ss. to be given as needed, quinine gr. viij. daily, and liquid diet.

23d. Feels very well; bowels regular; no pain; appetite is good. Temperature has ranged from 98° to 98½°.

Case VI.—Diarrhœa with typhoid prostration, M. W., æt. 19, born in Germany, single, laborer; admitted April 24, 1882; discharged April 29, 1882, cured. Has generally been very healthy. Came to this country eight months ago; two weeks ago he began working at a sugar refinery; about a week ago he was taken with diarrhœa, which has since become worse; has not had any nausea or pain.

Upon admission patient is slightly prostrated; face flushed; temperature also subnormal, 98°; tongue slightly furred, moist; pulse slow; appetite poor; bowels loose. Examination of heart, lungs, and urine gives negative results. Ordered Hope's camphor mixt. f℥ss. to be given as needed.

29th. Diarrhœa has been checked, appetite has improved, and he seems very well again.[1]

[1] In a conversation with one of the proprietors of the sugar refinery in question, the opinion was expressed by him that the symptoms in the above cases were caused by the men drinking large quantities of sweetened water.

REMARKABLE CASE

OF

SACCULATED OR OF CIRSOID ANEURISM OF THE SECOND INTEROSSEOUS BRANCH OF THE DEEP PALMAR ARCH TREATED BY EXCISION.

WITH EXHIBITION OF THE SPECIMEN.

By

JOHN B. ROBERTS, M.D.,

LECTURER ON ANATOMY AND ON OPERATIVE SURGERY IN THE PHILADELPHIA SCHOOL OF ANATOMY.

[Read May 3, 1882.]

The specimen which I exhibit this evening and its accompanying history are interesting, I think, because of the extreme rarity of the condition. I know of no similar case reported; but I have not had an opportunity to search for such in medical literature, because the operation was performed only a few hours ago. The specimen is fresh, and is exhibited now before the appearances have been changed by any preservative fluid.

Dr. Charles H. Thomas requested me a few days ago to assist him, at an early date, in operating upon a tumor of the hand in a boy aged sixteen years. From his earliest childhood he had been under Dr. Thomas's observation, and had

had a small elongated tumor upon the *dorsal* surface of the first phalanx of the left ring-finger, while in the *palm*, at the junction of the bases of the middle and ring-fingers, was a larger swelling. These were considered masses of dilated veins, as they had a spongy feel, and at times showed a bluish color. There was no very definite connecting band of swelling between the dorsal and palmar enlargements. No special pain was experienced unless the parts were struck, and no marked growth occurred. Hence the child's mother was advised to have nothing done. As the boy grew, the hand and tumor increased, but held the same relative proportions. When the boy began work in a machine shop, the skin became thickened and soiled, and the bluish tint was no longer discernible.

About two months or less ago, the growths seemed to enlarge and to be accompanied by considerable pain, and Dr. Thomas advised the use of a compress in the palm and a bandage around the finger. This the boy wore at nights, and usually from Saturday to Monday morning when he returned to his work. Recently there was noticed pulsation in the palmar tumor and a lobulated feel; and Dr. Thomas feared that an arterial aneurism existed.

When I examined the boy last evening, I found on the back of the third finger a hard fibrous-like tumor, as large as a watermelon seed, with the long diameter corresponding to the length of the phalanx. In the palm was an illy defined swelling covered with thick skin, very sensitive to pressure, and occupying about the area of a silver half-dollar. No swelling was evident connecting the two tumors. On the ulnar side of the palmar mass moderately distinct pulsation could be felt, which quickly stopped when the radial artery was compressed at the wrist, but merely decreased in force when the ulnar was pressed upon with the finger. No pulsation was felt in the dorsal tumor.

The boy had severe pain even when no pressure was made upon the growth in the palm.

I gave it as my opinion that the growth was an arterial angeioma connected with the second interosseous branch of the deep palmar arch, having anastomoses with the digital branches of the ulnar artery. Dr. Thomas considered it possibly this, but probably a sacculated aneurism. His diagnosis has proved to be the more correct.

It was determined to employ the Esmarch elastic bandage, and to make a free incision over the tumor and dissect it out, whether it be angeioma or aneurism. As Dr. Thomas was disabled by a painful boil on his right hand, he requested me to operate. The boy was etherized and the elastic bandage applied.

I made an incision from a point a little in front of the superficial palmar arch to the commissure of the fingers, and came upon a mass of fat and small vessels, in the centre of which was a bluish nodule, resembling larger vessels containing blood not driven out by the elastic bandage. Keeping close to the skin, and going down to the sheaths of the flexor tendons, I dissected the mass free. Lying alongside of the palmar interosseous muscle going to the ring-finger (2d interosseous) we saw a comparatively large vessel which seemed to be the main feeder of the mass. I then extended my incision, making a straight cut along the side of the ring-finger, dissected up the skin, and enucleated the hard nodule lying on the back of the first phalanx. This seemed connected with the other mass by some fibres or small vessels, and both were removed as one piece. The wound was then plugged with dry muslin to stop the general oozing that occurred after removal of the bandage, and a tight bandage applied. No ligatures were required, because my incisions were made at a distance from the tumor.

Dissection of the palmar mass showed that I had removed a small body, about three-quarters of an inch in

diameter, containing clotted blood, and surrounded by adipose tissue and nerves. The tumor, when freed from other structures, as is seen on the table, consists of three lobules of rather unequal size, arranged somewhat as a trefoil. The largest one of them, which has been punctured, allows the escape of soft clot; this sac is about one-half an inch in diameter. The three sacs seem to be separate, because the head of a pin introduced into one does not pass into the others. The two smaller sacs or lobules are hard, as if the clot was old. One has been laid open, and shows a white centre, or nucleus, of cartilaginous consistence, surrounded by a layer of red clot. On the surface of this three-lobed tumor runs a nerve, which probably was the seat of pain from pressure, and parallel to it a small artery. Both of these become lost in the mass, at the upper end of the tumor, which was thought to contain the main supply of the aneurism, and around which a string was tied and left for identification.

The tumor from the back of the finger is hard, and on section shows an irregularly colored red surface. I believe the tumors, therefore, to be small sacculated aneurisms evidently allied to or identical with the variety called cirsoid aneurism. The one on the back of the finger and the two smaller lobules in the palm are undergoing cure by coagulation, induced in the dorsal one undoubtedly by the pressure from the bandage used at intervals during the last six weeks or two months.

If the diagnosis had been more certain as to aneurism, I believe that digital compression of the radial and ulnar arteries, or the use of an Esmarch elastic

bandage to the forearm, would have been proper treatment before excision was attempted.

The early period of life (about three years) at which the trouble was noticed primarily, renders it probable that the aneurisms were not originally traumatic.

The similarity to cirsoid aneurism is certainly very great, though there are some points which differ somewhat from the usual clinical history of these growths.

Two weeks after the operation the wound was closing satisfactorily by granulations.

[After the reading of the preceding paper:—]

Dr. W. W. KEEN called attention to the danger of using coagulating agents in such cases, and spoke of a case of traumatic origin he had seen in consultation, in which a few drops of Monsell's solution had been injected into the aneurismal sac, and gangrene had followed, necessitating amputation of the hand. He thought compression of the radial and ulnar arteries would probably have accomplished a cure, and the risks of an operation would have been avoided. With regard to the small tumor on the dorsal surface of the ring finger, he thought it unlikely to be an aneurism. Nothing short of a microscopical examination would determine its nature.

REPORT OF THREE CASES OF ABSCESS OF THE BRAIN.

By

J. T. ESKRIDGE, M.D.,

PHYSICIAN TO THE HOWARD AND ST. MARY'S HOSPITALS.

[Read May 3, 1882.]

Abscess of the brain is of sufficiently frequent occurrence to make the subject of interest to the general practitioner. The cases reported in this paper are all the more interesting because each, due to a different cause, was associated with dissimilar diseases.

The first case was associated with cancer in several of the other organs of the body; the second, with chronic meningitis; the third, with apparent facial erysipelas and pneumonia followed by acute meningitis.

Detailed cases of brain lesions lose half of their interest, and become almost valueless to students of cerebral localization, when unattended by accurate descriptions of the relations of pathological lesions to ganglia, fissures, and convolutions.

I wish to acknowledge the services of my friend Dr. Charles K. Mills, who took an active part in the post-mortem examination of Case I., and rendered me valuable assistance in accurately locating the lesions in the first two cases.

CASE I. *Two Abscesses of the Brain.*—1. The larger one, involving the centrum ovale of the postero-frontal and parietal lobes, deeper portions of the gray matter of the anterior parietal convolution, and superior parietal lobule; 2. The smaller, the anterior two-thirds of the lenticular nucleus, and the adjoining portions of the external capsule and claustrum.

September 8, 1881, Mrs. D., æt. 54, born in Germany of a family several of whose members had died from cancer, had been a sufferer from headache fourteen years. She had a large frame and carried considerable fat. Excepting the head trouble, she had enjoyed excellent health until two years before, when she noticed a small tumor involving the left mammary gland. This proved to be cancerous, grew gradually, and was removed by a surgeon one year after its first appearance. There had been no return of the growth, although some pain had been complained of in the region from which it had been excised.

Her head pain had greatly increased in severity during the last few months.

In the early part of July, 1881, the left arm began to feel weak, and ten or twelve days later, the left leg was similarly affected, but to a less extent. About this time she went to the country for her health. Continuing to grow worse, she returned home during August, three weeks before I first saw her, and was able to walk from the street cars to her house, a few hundred yards distant. She was then treated by a homœopathic physician for a few weeks, when I was called in, and made the following notes:—

The left leg was almost completely paralyzed, the muscles feeling soft and flabby. The left arm was entirely paralyzed. The muscles of the left side of the face were slightly paretic. The right pupil was normal in size, left slightly dilated, and responded imperfectly to light. There was no paralysis of the muscles of the tongue, palate, or pharynx. The tongue

was slowly protruded in the median line, but when it was held in this position a few seconds it became tremulous, a vibratory tremor taking place in individual muscular fibres. Muscular power was impaired in the right arm and leg, but co-ordination was perfect. She had had no convulsions, and there were no muscular twitchings.

With the exception of a slight tendency to drooping of the upper lid of the left eye, the ocular muscles seemed to be acting well and in harmony.

Sensation was unimpaired on both sides of the body.

Special Senses.—Vision was equal in each eye, and she read Snellen No. 8, held at about fourteen inches. The ophthalmoscope revealed nothing beyond a slight haziness of the right retina. Taste, smell, and hearing were nearly normal, being well preserved for a woman of her age. Speech was unaffected.

The tongue was moist and slightly coated, appetite poor, bowels constipated, not having been opened for several days. The urine was high colored, sp. gr. 1025, but free from albumen. The bladder was tolerant and acted voluntarily.

She was restless, did not sleep well, and complained bitterly of pain in her head. The pain was usually dull. Its seats of intensity were at the top of the head and above each temple, being worse on the right side. She was intelligent, her mind was clear, but being under the impression that her cancer would return, she worried, had become emotional and imaginative.

Temperature (left axillary) 99°; pulse 76, full, soft, and regular; respirations 24 per minute.

September 10, two days later, during my absence from the city, she was seen for me by Dr. Mills, who reported her to be about in the same condition.

September 12. She was not so intelligent, worried more, and refused to eat. The feet were extended and inverted, the balls of the great toes lying in contact with each other. The internal and posterior muscles of the legs were so firmly contracted that it required considerable force on the part of

the attendant to evert and flex the feet, and the effort gave her decided pain. The muscles of the left side of the face were almost completely paralyzed. Swallowing solid or semi-solid substances was so difficult that she refused to eat.

Temperature, right axilla 96°, left axilla 99.4°, right elbow 95°, left elbow 96.7°. Pulse 86; respirations 26 per minute. Respiration was largely of the abdominal type. After taking citrate of magnesium, her bowels were opened once yesterday, being the first time in about a week.

Sept. 13th. At 11 A.M., the temperature of the room being 80°, after ascertaining her body heat, as indicated in each axilla, I made observations on her cerebral temperature, applying the thermometer to most of the stations used by Broca, Gray, and later by Mills. On the present occasion, the surface temperature of the head was taken at eight different stations, on a subsequent one, at two, and still later, at eleven. Hick's surface thermometer was employed and allowed to remain in place ten minutes. On each occasion the temperature of the room was ascertained, and that of each axilla noted. The observations were made by myself between the hours of 11 A. M. and 2 P. M. The selected stations were as follows: 1. A middle frontal station in the centre of the forehead. 2. A right anterior frontal station, over the right eye, an inch and a half above the brow and the same distance from the median line of the forehead. 3. A left anterior frontal station, similarly located on the left side. 4. A right posterior frontal station, near the external angular process of the frontal bone. 5. A left posterior frontal station, corresponding in position on the left side to that on the right. 6. A right parietal station, just above the right ear. 7. A left parietal station, just above the left ear. 8. A right Rolandic station, situated on or near a line drawn over the top of the head from the right to the left auditory meatus, and from two to three inches above the top of the right ear. 9. A left Rolandic station, situated similarly to that on the right. 10. A right occipital station, one and a half inches to the right of the centre of the occipital region. 11. A left

occipital station, one and a half inches to the left of the centre of the occipital region.

I shall now give the temperature records in connection with the history of the case, and further details under the head of remarks.

Temperature in right axilla 97.9°, in left axilla 98.4°. Pulse 104, weak, and unsteady; respirations 24 per minute.

Surface Temperature of the Head.

Right Side.		Left Side.	
Anterior frontal station,	97.3°	Anterior frontal station,	97.7°
Posterior " "	97.8°	Posterior " "	98.1°
Parietal "	97.1°	Parietal "	97.9°
Rolandic "	96.3°	Rolandic "	96.8°

She was unable to void her urine for the first time to-day. Thirty-two ounces were drawn off by the catheter. From this time to the date of her death, just two months later, the catheter was daily required to relieve the bladder. She had not eaten anything for two days. Headache was excruciating. One-half of a grain of morphia was required each night to produce sleep, bromide of potassium making her wild and delirious. During the latter part of the day she frequently talked incoherently.

September 15th. The temperature of the left Rolandic station was 5.5° higher than that of the right. The intermitting character of the respiration formed a very interesting feature of the case at this time. There was no gradual rise and fall of the respiratory movements as seen in the typical Cheyne-Stokes' respiration. There were twenty-eight shallow respirations per minute. After four or five short and rapid respirations there was an interval of rest of eight or ten seconds, then after about the same number of respirations, came another interval of rest. During the next week, the pain in the head became more intense, and localized in

the top of the head and right temple. The bowels failed to respond to various purgatives and enemata.

September 22d. The muscles of the left side of the face showed but little loss of power. The pupils were of equal size. The left leg, as well as the left arm, was now entirely paralyzed. The muscular strength on the right side did not fail faster than is usual in one whose general vitality is being lowered.

The conjunctiva of the right eye was congested and watery, the sight being but little impaired.

Temperature, Right axilla 97.6°; left axilla 98.2°; pulse 68; respirations 12 per minute. Respiration was still of the abdominal type. The movements of the abdomen preceded those of the chest, and formed with them a see-saw like, or to-and-fro motion, the chest apparently not expanding until the end of inspiration and beginning of expiration.

September 23d. Temperature of room, 82°; of right axilla 98.9°; left axilla 98.2°; pulse, 72; respirations 9 per minute.

Surface Temperature of the Head.

Right Side.		Left Side.	
Anterior central station, 96.3°.			
Anterior frontal station,	95.9°	Anterior frontal station,	96.2°
Posterior " "	95.6°	Posterior " "	97.6°
Parietal "	97.6°	Parietal "	98.1°
Rolandic "	97.0°	Rolandic "	96.8°
Occipital "	96.9°	Occipital "	98.2°

The pulse, although only 72 per minute, was very weak, but regular in time and volume. The intermitting character of the respiration was more marked on this than on the preceding day. Three or four respiratory movements were followed by an interval of rest, lasting from twelve to fifteen seconds. The number of respirations did not exceed four in succession, nor were they, at any time, less than three. The interval, or rest, was not longer than eighteen seconds, nor was it less than twelve. The respirations, which were largely abdominal, had to be counted for two minutes to

obtain the average number per minute. If the movements of respiration were looked for at the beginning of an interval, none were seen during the first quarter or third of the minute, and only three or four during the second quarter or third; whereas, during the latter part of the minute, there might be eight, six, or only three respirations. Thus:—

Inter.	Respirations.	Int.	Res.	Int.	Res.	Int.	Res.	Int.	Res.	Respirations.
	4		4		4		4		4	= 20
15s.	9s.	15s.	9s.	15s.	9s.	15s.	9s.	15s.	9s.	= 120 sec.

It will be seen by observing the above tabular view that, if the respirations had been counted during one minute only, eight might represent the breathing frequency per minute; and if those paroxysms of respiration, if I may so term them, had been limited to three respiratory movements each, as they sometimes were, six, instead of ten, of the tabulated form, could have been set down as the number of respirations. This character of breathing, sometimes slightly increasing in frequency, continued until a few days before her death, when it changed to the so-called Cheyne-Stokes' respiration.

Sighing rarely occurred. The pupils were normal. The bowels had not been opened since the 11th instant, notwithstanding various purgatives and enemata had been given. Croton oil had been given by the mouth until it had produced griping pains, and an emesis of glary mucus, yet it seemed to have no effect on the bowels. The patient lived from the 11th of September to the 13th of November without having an evacuation from the bowels. During this period of two months and two days, or about nine weeks, she neither suffered from nausea, vomiting, nor distension of the bowels from gas or feces. Her diet for more than two months just preceding her death was almost exclusively of a liquid nature, she rarely taking any semi-solid food.

September 28th. During the last few days the face had been turned to the right, and the head drawn toward the right shoulder. At this time she complained of great pain and soreness in the right and posterior portions of the neck. The parts were hard, swollen, and painful to the touch. An

attempt, on my part, to straighten the neck greatly augmented her suffering. Pain was also complained of in the right eye and ear. The former was inflamed, and almost useless as a visual organ, and from the latter there was a constant discharge of yellow pus. The ocular inflammation involved more especially the conjunctiva and cornea. An ophthalmoscopic examination of the right eye was unsatisfactory on account of the hazy condition of the cornea and the uncontrollable movements of the eyes. The watch was not heard by the right ear, except on firm pressure.

October 3d. She was blind in the right eye and deaf in the right ear. The discharges from the eye and ear continued.

October 13th. She remained quiet during the day, but required two grains of morphia to induce sleep at night. The hearing in the left ear varied; at times she heard tolerably well with this ear, and at others she was almost deaf. No improvement in the right eye and ear. A semi-choked disk appearance was noticed in the left eye, and vision was nearly gone. The left eye showed no evidence of external inflammation. No discharge was noticed from the left ear.

November 11th, 5 P. M. Temperature, right axilla 95.5°; left axilla 98.2°; pulse 104; respirations, 28 per minute.

The right side of the body felt quite cool. The breathing had changed from an intermitting to an irregular character, and constituted what is known as the Cheyne-Stokes' respiration, except that there was no lull, during which the hand could not detect feeble movements of the thoracic wall. The respiratory movements gradually became shorter until they were almost imperceptible, then they gradually lengthened until a full inspiration was taken with considerable effort, followed by a blowing noise. Every paroxysm, or rise and fall of the respiratory acts, included about eighteen respirations. She neither saw nor heard, and was no longer able to speak or swallow. The saliva was wiped from the mouth by the attendant, or she made an awkward and ineffectual effort to do it herself, showing that she was probably not

entirely unconscious. Her urine was passed involuntarily. No morphia had been required during the last few days. The neck remained in about the same condition that it was on September 23d. The muscles of the left side of the face were paralyzed, the mouth being drawn well over to the right side.

She died, exhausted, November 13, 1881, at 12.30 P. M.

Post-mortem examination was made three hours after death by Drs. Mills, Bissey, and myself.

Brain.—The venous sinuses and arteries were carefully examined, but were found to present nothing abnormal. No lesion of the membranes was discovered. The pia mater was unusually transparent (almost bloodless). The optic nerves were normal in appearance, and no perceptible lesion was detected in the other cranial nerves. No effusion. Entire surface of brain examined through the transparent pia mater before its removal, and no pathological condition was discoverable. On stripping the pia mater from the right hemisphere, the external surface of the convolutions was still found to be intact. Before completing the process of removal of the pia mater from the right hemisphere a large quantity of a dirty grayish-white liquid suddenly escaped

FIG. 1.

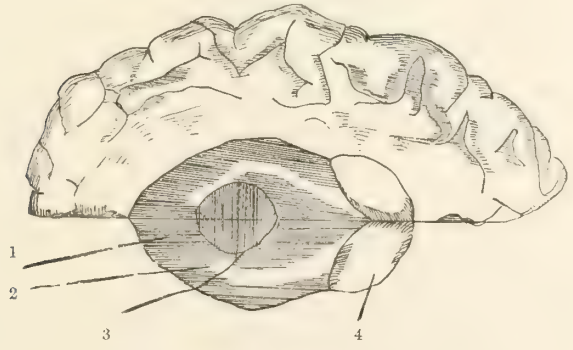


FIG. 1.—Abscess (shaded) in the right lenticular nucleus, as seen by a longitudinal incision. 1. Caudate nucleus. 2. Internal capsule. 3. Lenticular nucleus. 4. Optic thalamus.

through a small opening at the bottom of a secondary fissure just in front of the upper extremity of the fissure of Rolando.

The right lateral ventricle was laid open by an incision through the lateral border of the corpus callosum. The surfaces of the intra-ventricular ganglia were healthy in appearance. The floors and roofs of the cornua showed no lesion. A longitudinal incision was made through the middle of the head of the caudate nucleus and the optic thalamus,

FIG. 2.

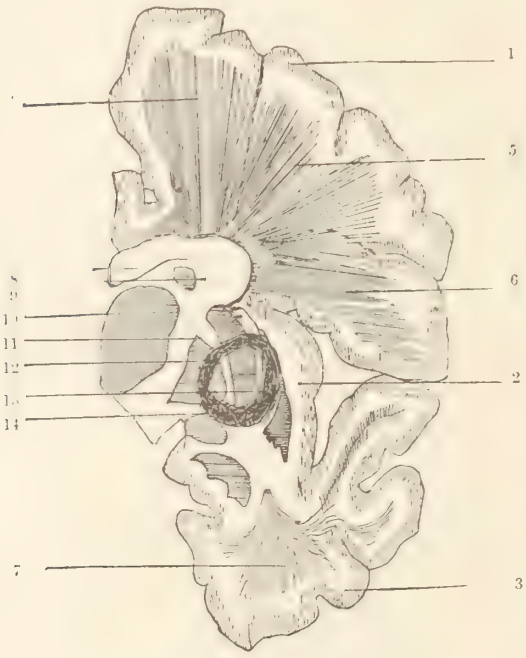


FIG. 2 (from Ferrier).—Abscess (shaded) as seen by a vertical transverse section (frontal). Frontal section. 1. Ascending frontal convolution. 2. Insular lobule. 3. Sphenoidal lobe. 4, 5, 6. Superior, middle, and inferior frontal fasciculus. 7. Sphenoidal fasciculus. 8. Corpus callosum. 9. Caudate nucleus. 10. Optic thalamus. 11. Internal capsule. 12. Lenticular nucleus. 13. External capsule. 14. Claustrum (Vormaner).

deep enough to pass through these bodies and the lenticular body, just to the inner margin of the insular and sphenoidal cortical substance (gray matter). This incision (Fig. 1) exposed a small isolated abscess about one inch in length, three quarters in depth, and one-third of an inch in width, involving the following parts from before backwards; the anterior two fifths of the lenticular nucleus, and the adjoining portions of the external capsule and claustrum. The caudate nucleus, optic thalamus, and the entire internal capsule (Fig. 2) were not changed in appearance. The posterior three fifths of the lenticular nucleus and a very narrow strip of its anterior two-fifths above the abscess were intact. Vertico-transverse sections through the pre-frontal and occipital regions showed no lesions. Further transverse sections

FIG. 3.

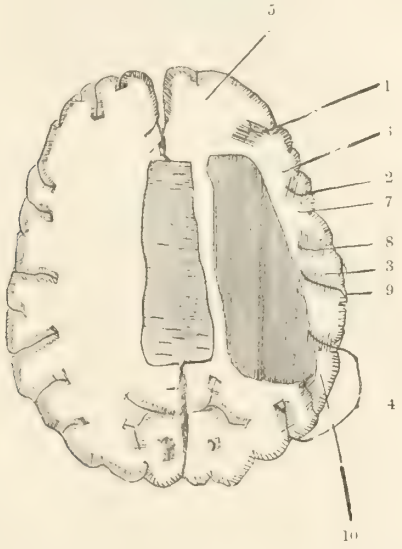


FIG. 3. Abscess (shaded) as seen by a longitudino-horizontal section on a level with the corpus callosum. 1. Upper frontal fissure. 2. Vertical frontal fissure. 3. Fissure of Rolando. 4. Interparietal fissure. 5, 6, and 7. Upper, Middle, and Lower frontal convolutions. 8 and 9. Anterior and Posterior central convolutions. 10. Parietal lobules.

revealed a nodulated mass and large abscess of the centrum ovale of the postero-frontal and parietal lobes.

The nodule (Fig. 4) was about the size of a large hickory-nut. It involved the white matter and the under portion of gray matter at a point corresponding externally to the superior parietal lobule or convolution, a little anterior to the middle of the inter-parietal fissure. Its anterior extremity just grazed the middle portion of the gray matter of the ascending parietal convolution. The nodule was apparently firm. The abscess (Figs. 3 and 4) extended on all sides of

Fig. 4.

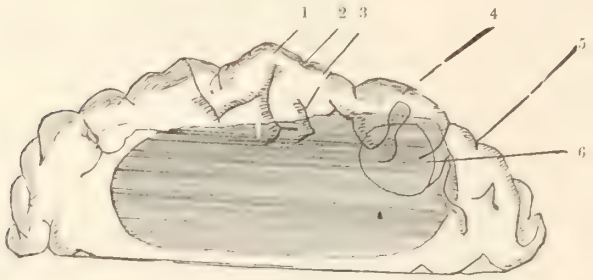


Fig. 4.—Abscess (shaded) as seen by a vertico-longitudinal section made a little externally to the basal ganglia through the cortical substance and centrum ovale of the right hemisphere. 1. Ascending frontal convolution. 2. Fissure (upper end) of Rolando. 3. Ascending parietal convolution. 4. Superior parietal convolution. 5. Parieto-occipital fissure. 6. Nodule.

the irregular body, and the exact limits of the former were determined by transverse sections to be as follows: Its anterior extremity reached a point in the centrum ovale corresponding to a plane, just in front of the caudate and lenticular bodies, externally through the anterior halves of the first, second, and third frontal convolutions. Its posterior extremity reached a point in the centrum ovale of the parietal near the plane of the parieto-occipital fissure. The abscess in its anterior half involved white matter only, but close up to the gray substance. Its posterior portion involved the

white matter, also the inner part of the gray substance of the adjoining convolutions. The posterior one-fourth of the abscess involved the gray matter to the greatest extent, and approached near the surface at the most posterior part. It had, therefore, destroyed the centrum ovale directly in relation with, *a*, the posterior parts of the first and second frontal convolutions; *b*, the middle regions of the ascending frontal and ascending parietal convolutions; *c*, the middle and lower portions of the superior parietal convolutions; *d*, the upper portion of the inferior parietal convolution. The gray matter destroyed was chiefly in the anterior parietal convolution and the superior parietal lobule. The entire length of the abscess was between four and five inches.

The left hemisphere showed no pathological lesions.

Thorax.—Heart and large vessels were normal in appearance. The apex of the left lung contained four cancerous nodules, varying in size from that of a lima bean to a horse-chestnut. The apex of the right lung was the seat of a nodule about the size of a hickory-nut. The middle and lower portions of the lungs were anæmic. The pleuræ were normal, except at the apices of the lungs, where slight adhesions were formed over the nodular growths. Slight effusion in the left pleural cavity.

Abdomen.—The uterus, ovaries, and ligaments of the womb were the seat of fourteen cancerous nodules, most of which were not larger than a hickory-nut. The liver, spleen, kidneys, and pancreas were anæmic, but free from cancerous growths. The stomach was empty, pale, and contracted, but no induration of its walls was discovered. The upper and lower bowels contained a slight amount of fecal matter scattered throughout their extire extent. The contents of the bowels were soft. No evidence of intestinal obstruction could be detected. The rectum was empty.

Microscopical Appearances.—The nodule in the brain was composed of bloodvessels, brain-substance, extravasated blood, and blood-pigment No cells of a suspicious character were seen.

Remarks.—The abscesses cannot be attributed to the presence of a cerebral tumor. The history of the removal of a malignant growth from the left breast one year before, naturally suggested to me the probability of a similar one in the brain. No local interference with the venous or arterial circulation of the brain was detected at the autopsy. As causative agents, apparently nothing beyond a profound lowering of the vital forces and the blood infection attendant upon the presence in the system of several cancerous masses were found. If in these lies a sufficient cause, I am at a loss to know why abscess of the brain is so infrequently found in connection with cancer of other portions of the body, or why it is not more generally associated with general anæmia.

Remittent respiration is referred to by Hughlings Jackson (Reynolds's *System of Medicine*), although he does not give it that name, and Cheyne-Stokes' respiration by nearly every recent author on nervous diseases, but I find no allusion to the peculiar intermittent variety, found in this case in which three or four respiratory movements of equal length and depth were followed by distinct intermissions varying in length from fifteen to eighteen seconds.

Just before the patient's death, when the respiration approached the Cheyne-Stokes' character, there was no period or interval of several seconds' duration, during which the respiration intermitted, but the movements were continued regularly, increasing or lessening as the efforts at respiration reached the maximum or minimum.

One or more cases with respirations differing from the Cheyne-Stokes' similarly to this may be found

described in a joint paper presented at the Northern Medical Society of Philadelphia, by Drs. Mills and Ott. (*Phila. Med. and Surg. Reporter*, July 2, 1881.)

The abdominal respiration noted in the clinical history may be imitated by any one in health by first voluntarily expanding the abdomen, then allowing the chest to be inflated subsequently, the expiratory movements taking place in the order of time, in which the inspiratory were effected. It differs from the form of abdominal breathing referred to by Hughlings Jackson in connection with cerebral hemorrhage. It is a grave omen, and is most commonly found in cases of great prostration as in typhoid fever and shock, or it is associated with certain brain diseases attended by a similar condition of the vital forces.

The inflammatory changes that occurred in the right eye and ear with the subsequent loss of vision and hearing in these organs cannot be accounted for by any appreciable lesion in the pons, medulla, or at the base of the brain adjacent to the optic nerve. The well-known experiments of Waller, Bernard, and Brown-Séquard, on the effects of stimulation and paralysis of the cervical ganglia of the sympathetic nerve aid us in attributing the trophic changes found in the eye to the local trouble present in the right side of the neck, which preceded the affection of the organs of the special senses. I regret that we failed to examine the indurated tissues of the neck, and to ascertain the condition of the sympathetic nerve and ganglia in this region.

The general and cerebral thermometric records of this case present some points of interest.

General Thermometry.—According to the physio-

logical experiments of Hitzig, Eulenberg, and Landois (quoted by Mills, *Phila. Med. Times* for Jan. 18, 1879), the heat-controlling centres are situated in the cerebral cortex of the motor regions around the fissures of Rolando, especially in the ascending convolutions. Usually, but not constantly, destruction of this area gave rise to increased, and irritation produced lessened temperature in the extremities of the opposite side of the body. In the present instance the large abscess involved to some extent the supposed thermic area of the right side. Comparative observations of each axillary temperature were made on seven different occasions, and were as follows:—

	Right Axilla.	Left Axilla.
Sept. 8 . . .		99°
" 12 . . .	96°	99.4°
" 13 . . .	97.9°	98.4°
" 14 . . .	97.9°	98.2°
" 19 . . .	97°	98°
" 22 . . .	97.6°	98.2°
" 23 . . .	98.9°	98.2°
Nov. 11 . . .	95.5°	98.2°

The temperature of the right or non-paralyzed side of the body only once exceeded that of the left, when the thermometer registered .7° higher in the right than in the left axilla. The temperature of the left armpit ranged from .5° to 3° higher than that of the right. During the first shock to the system produced by the abscess, and just before death, when paralysis was most complete, the difference between the two sides of the body heat was greatest, being 3.4° at one time, and about 3° at another.

Cerebral Thermometry.—I shall only refer to the

extended observations of Broca, Gray, and Maragliano, and Sepelli, on cerebral themometry in health and disease. Their conclusions are in harmony with each other, and their utility has been demonstrated by observations made on at least two cases of tumor of the brain, one by Dr. Charles K. Mills (*Phila. Med. Times*, Jan. 18, 1879), and the other by Dr. Landon Carter Gray (Hamilton's work on *Nervous Diseases*, 2d ed., page 22), who was the first in this country to investigate the surface temperature of the head in health. Dr. Gray found the average normal temperatures of the stations on the left side of the head to be for the frontal, .65° ; for the parietal, .85° ; for the occipital, .72°, higher than those of the corresponding stations on the right side. He gave as the average normal temperature of the right frontal station, 93.71° ; of the left, 94.36° ; of the right parietal, 93.59° ; of the left, 94.44° ; of the right occipital, 91.94° ; of the left, 92.66°. Variations of more than 1.5° he considered suspicious of disease at that point, and of more than 2° strong evidence of a pathological condition.

Comparing the results of the surface temperatures of the head made on the case reported at length in this paper with those reported by Gray and others, we shall find several points of difference. The temperatures were from 2° to 4° higher than those obtained on the heads of healthy subjects, although the axillary temperatures of this case taken at the same time were normal or subnormal. The temperatures at the several stations on the left side of the head exceeded those on the right by about .5°, which is nearly in accord with the normal difference. The tempera-

ture at the left Rolandic station was .2° lower than that of the right on one occasion only. With this exception, the temperatures on the left side were the highest. On Sept. 23d, when paralysis of the left side of the body was complete, the surface heat at the left posterior frontal station was 2°, and at the left parietal 1.5° greater than at the corresponding stations on the right side. Before complete paralysis had taken place in the left leg, there was nearly an equal increase of heat at the frontal and posterior stations; later, when the leg was powerless, the temperatures of the posterior stations exceeded their normal more than did those of the anterior. In the observations on cerebral thermometry in connection with brain tumors made by Drs. Mills and Gray, the stations nearest the growths, both of which happened to be on the right side, gave the highest average increase of temperature.

The area to which the occluded vessels in embolic hemiplegia are distributed is said to have a subnormal temperature.

I am not aware that the results of any observations of the surface temperature of the head made on persons suffering from abscess of the brain have been reported. No valuable conclusions can be drawn from observations made on a single case; especially is this so when the surface temperatures of the head are not more frequently taken than in the history of the one just reported. In health the head temperatures are from 4° to 6° lower than that of the axilla, being lowest posteriorly. In the present case with a normal or subnormal axillary temperature, the head temperatures were elevated from 4° to 6° above their nor-

mal, the occipital, in one instance, being the highest, equalling the axillary, and exceeding its own in health 6°.

Dr. Allan McLane Hamilton, of New York, has reported the results of his observations on a case of meningitis, in which the occipital temperatures exceeded those of the other regions of the head. (Ibid.)

It is doubtful whether the local temperatures of the head in abscess of the brain will aid us in locating the disease to any particular region, but if the results of the observations already made on cerebral thermometry in brain tumor be sustained by future investigations, the surface thermometer may help us in differentiating abscess from tumor of the brain.

In a case of brain trouble, in which the diagnosis is between tumor and abscess, a limited surface area of one side (especially the right) of the head with a temperature considerably elevated above the heat of the surrounding parts, would point to tumor; whereas, an elevation of the entire cerebral surface temperature would indicate abscess.

The direction of the destructive lesion in the brain, if we may judge by the progress of the paralysis, was from before backward, showing that the nodule occupied the region last involved, instead of the first, as would have been the case if it had been caused by an embolus, and had in its turn given rise to the abscess.

Case II. Abscess of the brain following chronic meningitis: sudden death.

On the notes of Dr. Dennis the history of the following case is based:—

Mr. G. S., æt. 56, German, shoemaker, served in the German army, and when a young man led an irregular life.

He presented no marks of syphilis, and no history of his having suffered from that disease could be ascertained. He had always been a moderate drinker of beer, but never indulged in the stronger alcoholic liquors.

He was a large, heavily built man, and with the exception of occasional headaches, had enjoyed good health until the latter part of the year 1880, when the loss of his wife, followed by other domestic unhappiness, seemed sufficient to dis-arrange an already ill-arranged nervous system. His temper had been irritable for many years, but now he became subject to frequent outbursts of passion, during which he would vent his spleen on any one who chanced to be near him. His memory became very treacherous, often getting confused in his business, and even at times being unable to remember the day of the week. His headache became constant and more severe.

During the spring and early part of the summer of 1881, he had what he called "rheumatic attacks," during which, while attending to his usual duties, he would be seized suddenly by a sharp pain in the course of the left sciatic nerve, followed by immediate paralysis of the left arm and leg, compelling him to sit down to save himself from falling. During the attacks he did not lose consciousness. The paralysis never lasted longer than a few minutes, power of motion returning and seeming to be as good as before the shock. The frequency of the hemiplegic seizures, two or three attacks often occurring weekly, depended upon causes giving rise to outbursts of passion. Sudden changes of weather were apparently associated with the nervous disturbance.

His symptoms remained unchanged until the latter part of October, when in a fit of anger, he fell to the floor, frothed at the mouth and became convulsed. No paralysis followed the convulsion, but the headache steadily increased, compelling him to take to his bed. As on this, so on every succeeding day during his entire illness, he remained in bed only a portion of the day.

The head-pain now constant was so severe that it prevented

continuous sleep, and from short naps which he would occasionally get, he would be awakened suddenly by violent exacerbations of pain.

He was attended by homœopathic physicians until November 12th, two weeks before his death, when Dr. Dennis was requested to take charge of the case. His temperature at this time was 99.5°; pulse 70 per minute, full and strong; respirations natural; appetite capricious, tongue coated, stomach intolerant, and bowels obstinately constipated. Urine scanty, high-colored, and free from albumen, was voided voluntarily until a short time before his death, when it was occasionally passed involuntarily. The skin was slightly jaundiced. He complained of occasional chills, and was very sensitive to changes in the atmosphere. Nocturnal delirium soon began. He was very restless, saw strange things and people in his room, and would occasionally rise during the middle of the night and awake the whole family, telling them that it was time to get up for the day. At times his mind seemed to be a blank, he could not tell the day of the week, and would frequently be unable to distinguish day from night. On other occasions his mind was clear, but during these lucid intervals he could converse intelligently and connectedly for a few minutes only, thought suddenly ceasing to be generated, when a vacant stare would be his only reply to questions which a few minutes before he had answered promptly and correctly. Impressions were persistently and obstinately adhered to, and any attempt at persuasion to the contrary was not calmly received. His headache, principally frontal and right-sided, was now sharp and lancinating, requiring hypodermic injections of morphia and atrophia to relieve it. The orbital and other branches of the fifth nerves were very sensitive to pressure. His temperature during Dr. Dennis's attendance, though usually above the normal, reached 100° Fah. only on two or three occasions. The muscles of the left arm and leg were decidedly weaker than those of the right, yet sufficiently strong in the leg to support his weight for a short time.

Turning in bed was difficult. When the left arm unaided was held at right angles with the body a decided muscular twitching began. Occasional muscular twitchings took place when the arm was passive.

He thus continued, gradually getting worse, until November 25th, about five weeks after his giving up work, when Dr. Dennis requested me to see him in consultation.

When I first entered the room he seemed bright, and conversed intelligently. He had not talked long, however, before he began to hesitate in his answers, appearing to have difficulty in understanding me, and being more puzzled in framing answers to my questions. Soon he was unable to form more than one or two words of a sentence, and finally uttered a meaningless "yes" or "no," or stared vacantly, when spoken to. Pupils were small and equal in size, brows were contracted, and he complained of great pain in his head. The eyes were very sensitive to light, making it extremely difficult to make an accurate or satisfactory ophthalmoscopic examination. The acuteness of vision and the condition of the other special senses could not be ascertained. The retinæ were found to be slightly hyperæmic, and the optic disks somewhat swollen. No muscle or groups of muscles were paralyzed, but those of the left arm and leg were weak, and an attempt at co-ordination produced a muscular tremor. The left side of the face was apparently less expressive than the right. The heart, lungs, liver, and kidneys showed no evidence of disease. Temperature was 100°; pulse, 68, and full; respirations 16 per minute.

An opinion was given that the man was suffering from chronic meningitis of months' duration, and that a recent complication of softening or abscess of the brain had taken place.

As Dr. Dennis had enjoined rest in bed, and was doing everything necessary for the comfort and welfare of his patient, no change in treatment was made.

The next day Mr. S. was feeling so comfortable that he got up, dressed himself, and without assistance went down stairs from the third to the first floor to dinner. After eat-

ing he was helped to his third story room. He expressed himself as feeling tired and sleepy, and lay down to rest. He was left alone about one hour, when one of his daughters looking in his room found him on the floor, where he had fallen in getting out of bed. The left side was now found to be entirely paralyzed. He was placed in bed and left alone. Shortly after this, when looked after, he was found asleep and snoring loudly. As his repose was supposed to be a natural one, he was not immediately disturbed. About 6 P. M. the daughter, thinking it strange that nothing was heard from her father, went into his room and endeavored to arouse him, but to her surprise found him dead.

Post-mortem examination thirty-six hours after death.

Brain.—The dura mater over the entire extent of its bony attachment, was inflamed and firmly adherent to its osseous envelope. At the base of the brain the external membrane was so closely attached that it could be separated from the bone only in piecemeal by the scalpel. The cranial nerves were apparently uninvolved in the diseased process. No abnormal quantities of fluid were found in the subarachnoid space. The bloodvessels of the pia mater were engorged, and the membrane was slightly adherent to the brain substance. The entire external surface of the brain appeared nearly normal, presenting no gross lesions of softening. Superficial portions of convolutions in the neighborhood of the greatest amount of inflammation were examined under the microscope, and found to be in nearly a normal condition.

An abscess was found on the right side involving chiefly the centrum ovale of the pre-frontal region, but also to some extent, in its middle part, the inner layers of gray matter of the first, second, and third frontal convolutions. On attempting to localize exactly the posterior limits of the abscess, it was found that a vertico-transverse section through the anterior lobe made just in front of the pre-central fissure exter-

Fig. 5.

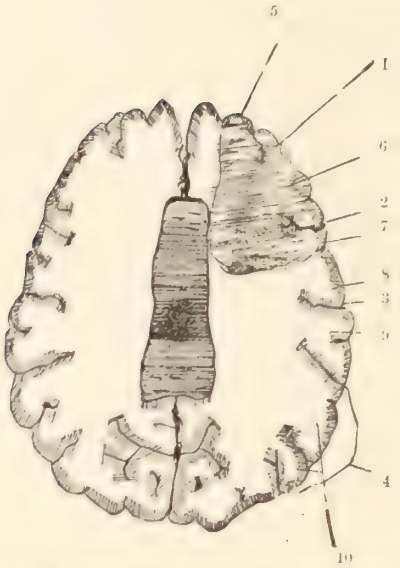


Fig. 5.—Abscess as seen by a longitudino-horizontal section on a level with the corpus callosum. The numbers represent the same parts as in Fig. 3.

nally, and striking the anterior extremities of the ganglia internally, just grazed its posterior limits. The abscess extended forward to within one inch of the anterior extremity of the lobe. Very few, if any, of the white fibres from the ascending frontal convolutions were involved. At the posterior and interior boundaries of the abscess a bloody nodulated mass two-thirds of an inch in its greatest dimensions was found. Other portions of the brain were apparently healthy. No emboli or thrombi were found.

The abscess did not communicate with the lateral ventricle, nor open upon the external surface of the brain to account for sudden death.

Microscopic examination of the nodulated mass and adjacent parts showed no new cell formation tissue; the nodule and the parts around exhibited nothing besides disintegrating brain tissue with its bloodvessels, and a few blood corpuscles.

FIG. 6.

FIG. 6.—Abscess (shaded) as seen by a vertico-longitudinal section made a little externally to the basal ganglia through the cortical substance and centrum ovale of the right hemisphere.

Remarks.—We cannot boast of our knowledge of the functions of the antero-frontal lobes, yet pathology, and later, physiology, have from time to time thrown some light on this obscure subject. The "crow-bar case" of Bigelow, the bullet wound related by Trousseau (quoted by Ferrier), the other cases mentioned, analyzed by Charcot and Pitres (reviewed by Ferrier, and quoted by Mills, *Phila. Med. Times*, Jan. 18, 1879), the tumor of the right antero-frontal lobe presented to the Pathological Society of Philadelphia, by Dr. C. K. Mills (*Transactions of Phila. Pathological Society*, vol. ix. page 106), and the lesion just described in connection with case second of this paper, in all of which the antero-frontal lobe was destroyed to a greater or less extent without any paralysis attributable to the lesion in this region, lead us to accept the conclusions of Charcot and Pitres, that motor disturbances of the slightest character do not necessarily follow lesions in the antero-frontal regions.

For the light physiology has shed upon the function of this region of the brain, I quote from Dr. Mills's article on tumor of the brain (*Phila. Med. Times*, Jan. 18, 1879): "Ferrier, *Functions of the Brain*, p. 230, in his experiments on the antero-frontal regions of the brain of the monkey, generally obtained negative results. Electrical irritation usually caused no special manifestations. Removal or destruction was not followed by any definite physiological results. The animals operated on, however, while not actually deprived of intelligence, had apparently lost the faculty of attentive and intelligent observations. When considering the hemispheres physiologically, Ferrier argues also that the power of fixing the attention and concentrating consciousness depends on inhibition. He shows that if the centres of inhibition, and thereby the faculty of attention, are weak, or present impulses unusually strong, volition becomes impulsive rather than deliberate."

It will be remembered that the patient in case second where the lesion was in the antero-frontal lobe presented symptoms in accord mainly with the physiological experiments of Ferrier. The man was emotional and passionate, and his power of attention was weak and of short duration. He seemed at times to lose will power. Only temporary paralysis was observed, and this always after some fatigue, which may have induced some restraining influence from other portions of the brain, or what is probably more correct, as some of the fibres connecting the motor region of the brain with the muscles of the arm and leg were destroyed, the remaining healthy fibres became exhausted from overwork.

Among the other interesting features of the second case are, the sudden death and the patient's ability at times to walk well, although at others the paralysis was so extensive that he was not able to stand.

That he was able to walk at all was apparently due to the fact, sustained by the post-mortem examination, that not all of the fibres through which motor impulses passed from the brain to the muscles of the leg were involved in the destructive process. When he was resting, a few fibres, for a short time, could do the work of a greater number.

If this view be correct, it will enable us to explain the cause of sudden death, although there was neither hemorrhagic apoplexy, nor bursting of the abscess into the ventricles, or on the surface of the brain. The unusual strain that he put upon his crippled brain a few hours before his death, in descending and ascending two flights of stairs, and in sitting up to eat his dinner, so exhausted his nerve centres that its effects came like a shock from a severe cerebral hemorrhage. The occurrence of the hemiplegic seizures may be accounted for, probably, by the sudden overloading of the congested vessels of the brain during his fits of anger.

Case III.—Abscess of the brain; gangrenous inflammation of the right sylvian artery, and occlusion of some of its branches; facial erysipelas; pneumonia in the left lung.

J. R., male, aged about 40, laborer in a sugar refinery, had been sick and delirious for several days, was brought into the St. Mary's Hospital during the past winter, in an unconscious condition. His previous history could not be ascertained. His respirations were rapid and abdominal; pulse

small, weak, and irregular; temperature not taken. No urine was obtained for examination. His face was swollen and of a dark erysipelatous hue. The swelling around the eyes was too great to admit of an examination of the pupils. The facial swelling and comatose condition prevented the detection of local palsies, if such existed. He did not regain consciousness, but died about six hours after his admission.

Autopsy eight hours after death.—The abdominal cavity was not opened. The entire left lung was in the second stage of pneumonia; the right lung, and the heart were in nearly normal conditions. Only slight quantities of serous fluid were found in the pericardium and right pleural sac; considerable fibro-serous effusion was found in the left pleura.

Brain.—The dura mater was slightly congested, but there were no evidences of decided inflammation in it. The pia

FIG. 7.

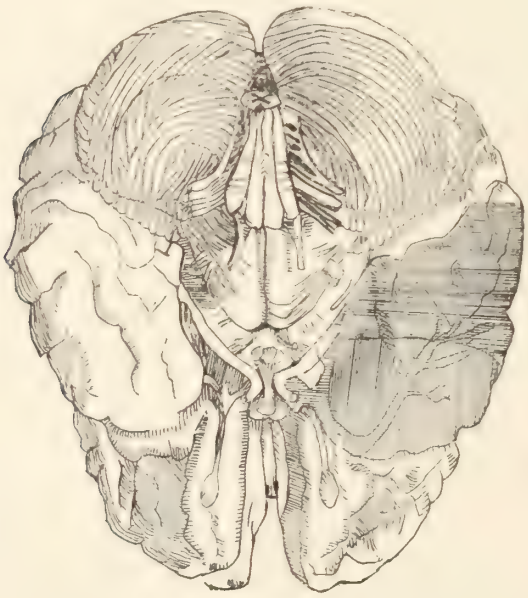


FIG. 7.—The dark portion of the right temporo-sphenoidal lobe denotes the seat of the abscess, the shaded portion of the same lobe the softened (superficial) surface.

mater covering the convex surfaces of both hemispheres, and that beneath the right was opaque. Its bloodvessels were highly engorged, and deposits of lymph and pus were found. At and just posterior to the right Sylvian fissure there was considerable quantity of pus, and sufficient lymph to so mask the structures at this point as to make them look like one mass of inflammatory products. The trunk of the Sylvian artery was almost black, its calibre lessened, but apparently not entirely obliterated. Some of its secondary branches, especially the lower ones from the parieto-sphenoidal and sphenoidal arteries, were occluded and seemingly gangrenous (Fig. 7). After carefully removing the pia mater, the convolutions and brain substance of the left hemisphere appeared nearly normal. The under and external surfaces of the right temporo-sphenoidal lobe from its anterior tip to its junction with the occipito-parietal lobules were softened to the depth of about an eighth of an inch. A superficial abscess or a mass of softened brain substance, about one inch in all its dimensions, occupied the anterior extremities of the first, second, and third temporo-sphenoidal lobules. The contents of the abscess were, in the centre, a watery fluid, surrounded by broken down brain substance and blood constituents, and on the outside, a thick layer of pus and other exudative matter.

The cranial nerves were but little encroached upon by the inflammatory deposits. Nothing special was noticed in the sinuses of the brain besides their blackened and engorged appearance. The ganglia were apparently normal.

The patient was not seen by me during life, and he was under the observation of Dr. Strittmatter the resident physician of the Hospital only six hours before his death. The circumstance of his death was investigated by the coroner, which necessitated great hurry in examining the brain and its membranes and sinuses. The consideration of the cause of the abscess and meningitis, and the nature of the erysipelas, I shall leave for those who may take part in the discussion.

[After the reading of the preceding paper, Dr. CHARLES K. MILLS said:—]

A careful examination of the cerebral sinuses might have thrown some additional light upon the third case. It was of great importance in all cases of abscess of the brain to investigate the condition of the sinuses of the dura mater. Abscess and thrombosis were not infrequently associated, and sometimes held to each other a causative relation. In the account of the condition of the patient (Case III.) it was stated that his face was swollen, and of a dark erysipelatous hue, the swelling around the eyes being too great to admit of an examination of the pupils or of the interior of the eyes. The erysipelatous appearance may have been the pseudo-erysipelas which accompanies some forms of cerebral thrombosis. The ophthalmic veins end in the cavernous sinuses. These veins have also a communication with the veins of the face. The facial vein, beginning on the anterior portion of the head, descends about the middle of the forehead, as the frontal vein, to the root of the nose, and then is continued downwards around the angle of the eye, under the name of the angular vein. This angular vein communicates with the ophthalmic vein. Hence it will be seen how swelling of those veins outside of the skull, which inosculate with the sinuses within, may become the most striking and positive signs of obstruction of the sinuses. Œdema of the eyelids and face, and congestion of the eyes, mean venous stasis in the parts drained by the ophthalmic veins. These phenomena were present in Dr. Eskridge's third case.

These three cases of abscess of the brain, well studied and described, constitute a valuable addition to the literature of intracranial diseases.

TENOSYNOVITIS: ITS CAUSES, NATURE, SYMPTOMS, AND TREATMENT; BASED UPON AN ANALYSIS OF FIFTEEN CASES.

By

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[Read June 7, 1882.]

TENOSYNOVITIS may be defined as an affection usually occurring in the forearm, and characterized by a peculiar creaking of the tendons as they move in their sheaths, depending upon a particular kind of strain to which the muscles belonging to these tendons have been subjected.

Cause.—The predisposing cause of the affection is the occupation of the individual, and in studying, therefore, fifteen cases, occurring in subjects of otherwise average health, the nature of their employment is worthy of special attention. In three of the fifteen, the disease occurred in men employed in a dye-house, whose work consisted in wringing the goods, which had been soaked in dye; in two, the patients were weavers, who throw the shuttle from side to side with the index finger of the right hand; one case occurred in a baker, from kneading bread; one in a boiler-riveter, from hammering; one in a car-driver,

from using the brake; one in an iron-moulder, from continued use of the shovel; one in a plaster-worker, from stirring plaster with a hoe; one in a washerwoman, from using a clothes-wringer; one in a laborer, who continued to work after receiving a severe contusion of the forearm from the fall of a heavy iron pipe; and one each in a rope-twister, a marble-rubber, and a painter.

In contrasting the above-named occupations with many others, requiring far more muscular effort, and giving employment to many more workmen than these, the idea suggests itself, that it is not the mere amount of strain to which the muscles and their tendons are put, that predisposes to the disease, but rather the kind of effort, which is of a tedious, continuous, monotonous sort. On the other hand, trades which would appear likely to furnish subjects for the disease more frequently than those which have been already spoken of, fail to do so. This, in some instances, can be explained. Gold beating, for example, where an eight-pound hammer is used almost uninterruptedly for five hours, and is carried from above the shoulder down to the level of the waist, would seem to contradict this view, as the disease is unknown to one of the largest gold-leaf manufacturers; a careful study of the movements of the operatives in performing this work, however, shows that the strain is not upon the muscles of the forearm, but rather upon those of the shoulder and arm; as the hammer descends simply by gravity and returns by recoil from the elastic block, composed of alternate sheets of gold and animal membrane, to a point where the biceps and deltoid muscles complete the elevation.

The exciting cause of the attack is usually the resumption of work to which the individual is thoroughly accustomed, after a shorter or longer interval, when he is out of practice, and when the parts involved in executing special movements have become less actively nourished; though in the case of the washerwoman, the clothes-wringer was used for the first time, and the rope-twister was doing work that was new to him. In the laborer the attack was of traumatic origin.

Pathology.—The means of determining the exact lesion in this disease are necessarily to a certain extent conjectural, but as the pain and crepitation are coincident in their onset and subsidence, as there is no impairment of motion after recovery has occurred, and as the parts under treatment regain their normal condition in a very short time, it seems highly probable that there is no true inflammatory process at all, certainly none extending beyond the stage of congestion, and that the creaking which exists is due to insufficient lubrication, with consequent dryness, not, as has been supposed, to exudation of lymph. Under rest and counter-irritation the congestion very soon disappears, the synovial surfaces pour out their proper fluid, and the tendons once more move smoothly and noiselessly in their sheaths.

Symptoms.—Soreness, amounting to positive pain upon motion or pressure along the course of the affected tendons, inability to use the part, and the presence of the peculiar creaking, which is communicated to the finger on palpation, are the symptoms which denote the existence of tenosynovitis.

Diagnosis.—From its common seat upon the dorsum

of the forearm, this affection may be mistaken for fracture of the radius. The history of the case, however, showing that there has been no blow or fall, as a rule; the quality of the crepitus, which is much softer and finer than that of fracture, and like that of cellular emphysema after fracture of the ribs, or that produced by rubbing two pieces of cloth between the fingers, and the way in which the crepitation may be elicited,—all leave little chance of error. The disease will not be mistaken for a strain of the muscle, if a careful physical examination is made.

Treatment.— From what has been already said, it will be seen that the disease is at once acute, painful, and disabling. It, however, yields, as a rule, readily to treatment; for the patient can seldom work more than a day after he is attacked, and finding that he exhausts the usual home embrocations, without relief, promptly seeks aid elsewhere; this enables the surgeon to institute treatment before an advanced stage is reached and permanent mischief done by a deposition of plastic matter. Absolute rest of all the parts concerned is the most important element in the treatment; a palmar splint, therefore, from the elbow to the tips of the fingers is applied, when the forearm is the part affected. Counter-irritation is next indicated, and may be employed in one of two ways. If the skin is red, a band one inch broad of tincture of iodine should be painted in an oval form around the area over which creaking is felt; while a lotion of lead water and laudanum is applied within this band. In cases where there is but slight creaking, and no redness of the skin, tincture of iodine may be painted directly over the diseased part, with-

out the employment of any lotion. The dressing is reapplied each day, until all pain, tenderness, and creaking have disappeared, which generally occurs at the end of four or five days. After this a roller bandage alone is continued, until the parts have regained their tone.

THE PRESENCE OF THE MICROCOCCUS IN THE BLOOD OF MALIGNANT MEASLES; ITS IMPORTANCE IN TREATMENT.

By

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[Read June 7, 1882.]

I PROPOSE to present for your consideration this evening the report of a recent epidemic in the Children's Asylum of the Philadelphia Hospital. The ward in which the disease first showed itself contained children between the ages of two and three years; some of them had been deserted by their mothers, and others had been placed there temporarily whilst the mothers were employed in duties about the establishment. For the most part, these children presented a fair appearance of health; they were seemingly well nourished, of good development, though probably they would have been classed as "strumous," if their large features and tendency to glandular enlargements and eczematous eruptions had received careful attention. Together with all children of this class living in asylums, they certainly presented an open field for the production of those complications that are usually such fatal attendants upon measles.

In order to save time, I shall in this paper simply narrate the history of the cases. The little patients were zealously cared for by Dr. H. E. Campbell, Resident Physician, to whom I am indebted for the record of the notes taken at each of my visits.

I shall also embody in this report the investigation undertaken by Dr. Henry F. Formad, now well known as the patient and thorough investigator of the microscopic appearance of the blood in diphtheria, associated with Dr. H. C. Wood, under the auspices of the National Board of Health. Dr. Formad examined almost daily the blood of each little patient, and together we noted the presence of micrococci in the malignant cases, and their absence in those of mild type. A record of these examinations was kept by Dr. W. A. Edwards, Assistant Pathologist, as also the records of the post-mortem examinations, and to him I am indebted for them. We entered this study with no preconceived views; the rapidity with which this exceedingly fatal epidemic came upon us necessitated careful study in order to attempt if possible to discover its cause. I present also some photographs which show well the appearance of the field in these cases.

Case I.—J. F., aged 2 years and 3 months, was taken sick April 12, 1882. There had been no cases of well-defined measles in the house at that time, although it was epidemic in the city. The child had a sore throat, some cough, with fever. The throat eruption was punctated and well marked. The child died in convulsions April 15, 1882.

Autopsy.—An ante-mortem (chicken fat) clot was found in the right heart, extending into the pulmonary artery. There was great systemic venous engorgement. A decided staining

was noticed upon the cadaver, especially about the temple, which caused the remark that the disease was probably one of the exanthemata. This child had been placed upon carbonate of ammonia, quinia, and digitalis; had had bromide of potassium, hot baths, and stimulants in small quantities.

Case II.—M. J., aged 3 years and 5 months, was taken sick April 9, 1882. A slight eruption on the face was noted April 11, 1882; the next day the eruption was complete over the entire body. In this case the eruption was irregular, was decidedly that of measles, but of malignant type. Still she did well until April 17, 1882, when suddenly the breathing became harsh, and pulse was rapid. At 4.30 P. M. had an attack of suffocation with rapid feeble pulse. There was general venous engorgement, the face and lips were purple; pulse growing more rapid. Hot baths with mustard and frictions were used, and carbonate of ammonia and digitalis given. It was suggested to bleed in this case, but the evidences of heart clot were so marked, and the child had exhibited so many evidences of malignancy at the onset, that baths were used instead. April 11, 1882, at 3 A. M. she was still growing worse despite treatment, and shortly after had a violent convulsion and died.

There was no autopsy permitted.

Case III.—A. G., æt. 16 months. Took sick April 15, 1882.

April 16th had prodromes of measles. The temperature and pulse ran as follows:—

16th. P. M. 102°.
17th. A. M. 101°; P. M. 103°; pulse 180.
18th. A. M. 102°; P. M. 103°; pulse 180.
19th. A. M. 101°; P. M. 102°; pulse 160.
20th. A. M. 101°; P. M. 102°; pulse 148.
21st. A. M. 100°; P. M. 100°; pulse 180.

In this case no defined eruption appeared on the body, although there was a decided papular eruption on the uvula and anterior half arches of the throat. Whilst showing this case to the class we noted streaks of grayish membrane

in the fauces. The child had decided bronchitis, and the voice showed that the laryngeal mucous membrane was also affected. This child was not at that time very ill. There was no question in our minds at the time that it was affected severely by the measles poison, that it was, in fact, another malignant case. It was carefully watched, the nourishment regularly given with quinia and iron daily, as was customary in all the cases.

On the 21st the breathing was noted as peculiar (I shall describe it hereafter). An emetic was ordered of ipecac. fearing an accumulation of mucus from the bronchitis present, and also carbonate of ammonia and digitalis and hot foot-baths were given. The child's intelligence seemed good, and it will be noted that the temperature was but 100°, while the pulse was 180.

At 8 P. M. a second attack of suffocation occurred, and the child died in violent convulsions. The venous engorgement was very marked.

Autopsy.—A. G., baby, age 1 year. Post-mortem examination held ten hours after death.

Heart. Left side and valves all normal.

Right side. A large ante-mortem clot filling the cavity of the right ventricle, and extending into the auricle; a clot was also seen in the pulmonary artery.

Lungs. Left, normal.

Right lung. At the base of this lung the lesions of pulmonary congestion were seen, especially where the lung approximates the diaphragm.

Intestines. Slightly congested and hyperæmic.

Mesenteric glands. Enlarged and infiltrated by simple congestion.

Kidneys Normal.

Blood. Taken from the heart cavity as soon as it was opened and examined showed micrococci in the liquor sanguinis and *in the white blood corpuscles.*

Case IV.—J. F. McH., æt. 23 months. This child had a typical attack of measles. The case was shown to my ward

class several times throughout its course, the eruption was studied carefully in all of its details, and my friend, Dr. John M. Taylor, obtained for me an excellent representation of the measles eruption in water colors from this case. I refer especially to these points as evidence that the epidemic was one of measles; the cases heretofore were so irregular as to leave room for doubt to those hearing the recital of their histories. The eruption was rapidly disappearing, and desquamation had set in. April 21st the bronchitis seemed to be aggravated, the respirations were 36, and expirations seemed unusually prolonged. The breathing was noisy; the heart's action was rapid, pulse 148. Suddenly, in the evening, an attack of suffocation came on, which was relieved by an inhalation of nitrite of amyl. On the morning of April 22d the heart was beating 168; the venous engorgement was very marked, the jugular veins standing out like whip-cords; the respirations were from 36 to 40, but the temperature was 99°. I saw the child at this time, and noted the gasping breathing, the feeble pulse, and the tumultuous action of the heart. There seemed to be capillary spasm, judging from the gasping breath and the imploring look which the child gave to all its attendants; and we at once gave an inhalation of *nitrite of amyl*. In a few moments it seemed relieved. The administration of carbonate of ammonia, digitalis, hot baths, etc., was rigidly adhered to. The child seemed comfortable until 4.30 P. M., when it had another attack of milder character, though longer duration, and in it finally died of convulsions. It was observed, says Dr. Campbell in his notes, that the convulsion was not as severe as in the previous cases.

Autopsy.—J. F. McH., post mortem made twenty hours after death.

Eruption not well marked.

Heart. Right ventricle contained a small ante-mortem clot. This clot was in the cavity of the ventricle, and did not involve the valves, either tricuspid or pulmonary. The left side of the heart was normal in every respect; contained no clot.

Lungs. Normal, with the exception of hypostatic conges-

tion at both bases. The pulmonary and costal pleura of the left side were inflamed and adherent in some places.

Trachea. Inflamed, and containing a tenacious mucous secretion.

Larynx. Inflamed and hyperæmic.

Liver. Normal.

Intestines. Peyer's patches and the solitary and agminated glands infiltrated and hyperæmic.

Mesenteric glands. Enlarged and infiltrated, they were about the size of a grain of corn.

Kidneys. Normal.

Spleen. Amyloid bodies enlarged until they presented almost the appearance seen in a tubercular spleen.

Blood. Taken from heart as soon as punctured. Micrococci were found in the liquor sanguinis and *in the white blood corpuscles, and they were mobile. In the corpuscles they were seen in great numbers in active movement of a vibratory or whirling character, and they appeared to have devoured the white cells.* No bacillæ were seen.

Case V.—J. McG., æt. 26 months. Ordinary case of measles. The eruption had disappeared on or before April 15th.

April 22. Child restless; marked bronchitis; cough paroxysmal upon waking, especially after excitement; mucous râles coarse, and fine throughout lungs posteriorly; throat congested, and saliva at times tinged with blood. At this date the breathing was noted as noisy. Pulse 144, respirations 32, temperature 100°.

April 23. Mucous rales becoming general, and not limited to areas as heretofore; pulse 152; respirations 34; temperature 101. In addition to the tonic and stimulating treatment, mustard poultices were applied to thorax and hot baths frequently given. Carbonate of ammonia was given now, gr. ij, every hour, and bisulphate of quinia by suppository, gr. ij, every three hours. In addition to this the child was given, as were all the others, milk and lime-water, beef tea, etc., at frequent intervals. We also in this case frequently

administered small doses of syr. ipecac to relieve the secretion, which was abundant and tenacious.

April 23. Evening. Pulse was rapid, 160 to 170; temperature 102½°; breathing becoming labored and gasping (fish-like); venous stasis was becoming more marked. Increased the whiskey to about one ounce a day. The attacks of suffocation continued paroxysmally; the jugular veins stood out like cords at times. *Nitrite of amyl* gave immediate relief, but relapse soon followed; it was always followed by free emesis, which seemed to be in itself beneficial. About midnight a severe paroxysm came on, and with it a convulsion in which the child died. After death the venous engorgement was more marked, and heart-clot had been suspected for some time before. This little patient was the first case whose blood Dr. Formad examined during life. The view of the fluid was photographed; *micrococci were found in great abundance, acting especially on the white corpuscles.* The blood was examined very shortly before the child's death, when the symptoms of heart-clot had been fairly established, and the case declared hopeless.

Unfortunately no autopsy was permitted in this case.

Case VI.—F. M., aged 2½ years. This case ran a course as did the others, and I will only occupy time with a description of the post-mortem appearances.

F. M., age 2½ years. Eruption well marked on mucous membrane of buccal cavity, not so on cutaneous surface.

Upon laying thorax open, lungs found to be anæmic, as far as arterial circulation was concerned, but dammed up with venous blood.

Heart. Normal in size and weight.

Right side contained a clot extending along the pulmonary artery for some distance; it was chicken fat in consistence.

Left side. Normal.

Spleen. Congested; weight four ounces.

Intestines. Along the small intestine could be seen a few Peyer's patches inflamed, and well outlined against the comparatively normal gut. The mesenteric glands presented a

very good example of enlargement and infiltration; they looked like so many peas scattered throughout the mesentery.

Liver. Normal.

Kidneys. Normal.

Brain. Not examined.

Blood. Taken from heart cavity as soon as it was open showed micrococci in the liquor sanguinis and *in the white blood corpuscles in abundance;* they were not mobile. A number of zooglæa masses were seen.

Case VII.—C. M., aged 2½ years. The eruption in the throat of this child was very well marked. A few crescentic points appeared in the temples, and the case rapidly developed malignant symptoms.

April 21. Slight grayish suspicious patches of membrane are seated in the throat. The child is hoarse, and there is much bronchitis.

April 22. Pulse rapid; respiration 28; breathing irregular. There is great general venous stasis, the skin dark and mottled.

Dr. Formad examined the blood microscopically, and found it full of micrococci. He took a specimen sample for photography. Prognosis very unfavorable, as the child has fluttering heart and gasping breathing. Hot baths had been used freely with no success; so also salicylic acid, which had been suggested early in the disease.

After consultation with Dr. Formad, the account of which I incorporate in the summary, it was concluded to give at once ℥ij of whiskey, and repeat it every hour; milk was continued as the only other food.

April 24. Pulse 144, temp. 101°, resp. 48. Circulation much improved. Venous engorgement relieved; breathing greatly improved. The child continued to improve during the day. At the end of the twenty-four hours it had taken 6 ounces of whiskey, and yet it showed no effects of alcoholism. At noon the pulse was 140, resp. 36; 6 P. M. pulse 132, resp. 32; 11 P. M. pulse 132, resp. 26, and regular, breathing easy, though somewhat noisy but not harsh.

April 25. A. M. temp. 98°, pulse 128, resp. 26; P. M. temp. 98°, pulse 108, resp. 24.

April 26. A. M. temp. 98°, pulse 96, resp. 22; P. M. temp. 98°, pulse 104, resp. 24.

The respirations remained regular, and the child continued to improve.

After the examination of the blood on April 30, owing to the relative increase of the white corpuscles, it was decided to give Fowler's solution of arsenic gtt. ij, three times daily. The large doses of whiskey were kept up for three or four days, and gradually diminished.

I give Dr. Formad's reports, which he kindly wrote out for me: —

Microscopic examination of the blood in the above case. (Examination made with a one-sixteenth immersion lens.)

Examination April 22, 1882.—Blood full of micrococci (sphero-bacteria), affecting many of the white blood corpuscles; also a large quantity of these fungi free and in various forms of grouping, mostly in zooglæa masses. White blood-corpuscles are in increased quantity; precipitation of fibrine excessively marked under the glass.

April 24. (Same case.) Micrococci present, but in diminished quantity; white blood corpuscles less affected; precipitation of fibrine less marked.

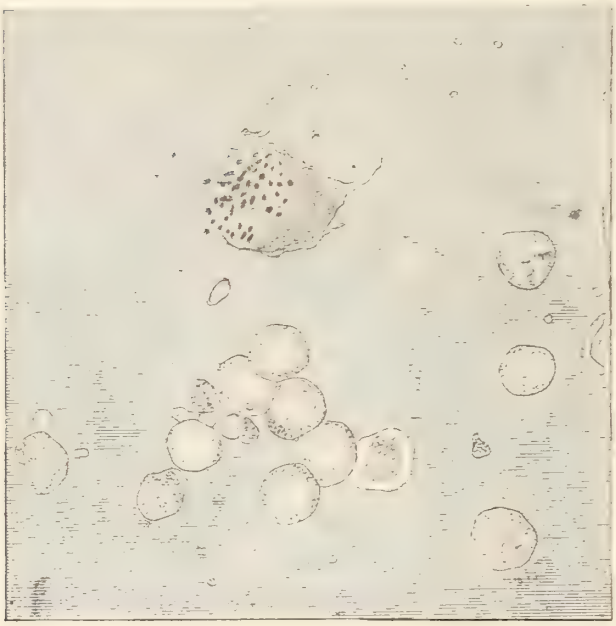
April 26. (Same case.) Micrococci very marked, yet principally in zooglæa masses and free in serum, but not affecting the white corpuscles; although the latter are in increased quantity; fibrine not noticeable.

April 30. (Same case.) Micrococci present; white blood corpuscles still in excess, but not affected by micrococci. Red blood corpuscles not readily forming rouleaux, having lost partly their bi-concavity.

May 3. (Same case.) Micrococci present in diminished quantity. White blood corpuscles diminishing in quantity.

May 7. (Same case.) Same as last.

May 18. (Same case.) Still some few micrococci present; blood otherwise appears normal.



Case VIII.—J. W., æt. 8 months. May 13. P. M. Eruption appeared on fifth day. The temperature ran as follows:—

May 13. A. M. ; P. M. 100⅗°.
May 14. A. M. 101⅘°; P. M. 102⅗.
May 15. A. M. 101; P. M. 103⅘.
May 16. A. M. 101⅖; P. M. 104.
May 17. A. M. 101; P. M. 102⅗.
May 18. A. M. 102; P. M. 103⅖.
May 19. A. M. 100; P. M. 103.
May 20. A. M. 103⅖; P. M. 102.
May 21. A. M. 105, death.

May 13. A fever mixture was given during the day. Quiniæ et ferri citratis gr. ij, every three hours.

May 18. The eruption fading, but leaving a purple stain and mottled appearance of skin. Catarrhal pneumonia or collapse probably exists, as the bronchitis is very extensive, the râles numerous, and subcrepitant. The blood examined under the microscope *shows micrococci in the blood corpuscles, but none free in the field. They are seen in great numbers.*

May 19, P. M. For the past two hours the child has been very restless, the breathing rapid and labored, and also spasmodic. No membrane on tonsils or fauces. Heart's action very rapid, venous stasis marked especially in the jugular veins. Gave hot baths (says Dr. Campbell's notes), and covered him with blankets, with some relief. Increased the whiskey to ʒij every hour.

In the evening gave an emetic. The child at night was breathing easier; friction sounds heard.

May 20. At times the strangulation would seem imminent. The venous engorgement increased, and the child died in convulsions on the morning of the 21st.

The post-mortem examination showed pneumonia and pleurisy with effusion.

The following eight cases were all taken sick at once, and I shall simply give a general statement of them for the purpose of especially calling attention to the case of W. L.

J. J., aged 4 years; W. L., aged 5 years; E. C., aged 3 years, catarrhal bronchitis; W. W., aged 3 years; C. B., aged 2 years, catarrhal bronchitis; J. W., aged 5 years; J. D., aged 5 years; L. K., aged 5 years.

Of these *eight*, seven presented severe but nevertheless typical examples of measles, and their blood was carefully examined by Dr. Formad and found normal. The case of W. L., who was taken ill at the same time as the others, showed from the onset a malignant tendency, giving a record such as I have already described. Dr. Formad gave me the following as the result of the examination of the blood in this case, and I had frequent occasion of examining it with him myself. Let me say that as soon as the presence of micrococci was established, the child was placed upon ʒij doses of whiskey every hour. Quiniæ et ferri citratis in citric acid, gr. ij. every three hours, friction to the extremities, and warm baths, with milk and beef-tea.

April 22. A few micrococci seen in the field.

April 26. Again noted.

April 30. Micrococci still present, white corpuscles increased, and marked precipitation of fibrine. *None were noted as having penetrated the corpuscles;* those that were found were simply in the serum. This child recovered, though every indication gave a very unfavorable prognosis.

In presenting this detailed report I desire to call especial attention to the following points, viz., the microscopic examination of the blood and the constant association of *micrococci* with the general manifestations of malignancy (a condition already well known), and the gradual but positive amelioration of all bad symptoms by treatment, which was directed to the micrococci as the *fons et origo* of trouble (this, I believe, for the first time exhibited).

It will be noted that the *post-mortem* examinations of these cases showed more or less simple pulmonary

congestion, and at times simple enlargement of the glands, but usually so circumscribed as to preclude the possibility of its being the immediate, or even remote cause of death. Again, the mode of death was peculiar: the fatal signs came on suddenly and with frightful intensity, the gasping breathing, the frantic efforts to obtain air (or really to aerate the blood), the imploring look, with consciousness not impaired, seemingly unduly acute, until the final convulsion or gradual cyanosis brought the end. The turgid veins, the occasional venous engorgement, the feeble pulse, and the fluttering heart pointed unmistakably to but one cause, the gradually forming right-sided heart-clot; and the *post-mortem* appearance, as these notes show, gave us a large, tough, chicken-fat clot, obstructing the venous circulation, firmly planted in the right heart and its tributaries, which was too often exhibited to raise a question. One of the earliest symptoms of this impending danger was undue rapidity of respiration. The child seemed to be doing well, its eruption irregular, probably incomplete, or dark and mottled, and in blotches, when attention would be called to the great rapidity of respiration with a peculiar gasping inspiration, fish-like in character. The other fatal symptoms would follow rapidly, and within twelve hours the child, despite carbonate of ammonia, warm baths, digitalis, etc., would die of heart-clot. What caused this?

In a short paper which appeared in the *Amer. Journ. of Med. Sciences* for January, 1882, I gave the experience of a number of cases of diphtheria, scarlet fever, and measles, and then attributed the condition to an increase of fibrine due to the rapid

tissue-changes and the malignancy of the type of disease, and urged the importance of pushing an alkaline treatment from the start.

The microscope has shown here that something more is associated with this condition.

The moment that symptoms of malignancy, viz., dark eruptions, ill-defined crescents, delayed and imperfect appearance of the eruption, with feeble circulation, high temperature, and pharyngeal false membrane appear, the examination of the blood showed *micrococci* in abundance in the field. They do not simply lie as impediments to the free passage of blood, though they most undoubtedly do this, and obstruct its passage in capillaries, but they surround the corpuscles, they enter the white corpuscles, and there develop with surprising rapidity, and finally cause some of them to rupture, and their contents will cover the field. Still, if they alone clogged the circulation in the capillaries, caused stasis in the lung, and thereby provoked an accumulation in the already enfeebled right heart, with blood having a tendency to coagulate, the cause of heart-clot alone would seem explained.

We find that they develop with activity, when the blood-current is retarded; hence we find them spread throughout the heart-clot itself, possibly at times having been here arrested by the obstruction to the flow caused by the lung congestion, known as a frequent complication of these cases, and finally aiding, by a mechanical cause alone, the deposition of fibrine that forms the clot. They do more. They act upon the white blood corpuscle, destroy it in all probability, or, at least, as one of the cases proves conclusively, prevent its change to red corpuscles, and thus the

oxygen-carriers being either destroyed or reduced in numbers with none to replace them, the tissues retain their detritus for want of carriers to relieve them, and another factor is added to increase mortality.

Granted, then, that the appearance of *micrococci* is coincident with symptoms of malignancy, we must assert that, whether their association be *post hoc* or *propter hoc*, they must have common cause; our treatment receives an impetus in a new direction.

I asked Dr. Formad what, in his experience, most readily checked the development of *micrococci* in his culture solutions obtained from erysipelas, diphtheria, etc.; he answered *alcohol*. Dr. Campbell at once withdrew carbonate of ammonia and digitalis from the treatment for the future, and gave whiskey. Five children had already died with the symptoms I have just described, and the sixth was exhibiting all the malignant symptoms, together with those which experience had taught us came from commencing heart-clot. The child had rapid gasping breathing, was becoming cyanosed, its heart was tumultuous, and the rapid pulse was growing weaker. The instructions were to give *three ounces of whiskey within the next twelve hours*, in frequent and small doses. The treatment was carefully carried out, and the child was saved. In this child *micrococci* were found in abundance in the blood, but none had penetrated the corpuscles, and for a long time the preponderance of white blood corpuscles was noted, which continued until gradually the blood became normal under the use of *arsenic*.

Again, let me illustrate another point. In one ward there were six cases at the height of eruption.

I carefully examined, with Drs. Campbell and Markoe, each case. One case was found to be of a malignant type. The child's right cheek was hardened and inflamed, and the mucous membrane showed that glistening surface so manifest in cancrum oris. The breath was fetid, there were cerebral symptoms, and a grayish exudation lined the fauces. We wished to test the microscope, so, without reference to any particular case, we requested Dr. Formad to examine the blood of all. In five the blood showed no *micrococci;* in one a large mass appeared in the field upon the first examination, and this one was the malignant case. This child was placed at once upon large doses of whiskey, and it was also given, in tonic doses, quiniæ et ferri citratis and citric acid.

The *vegetable* acids have also this remarkable effect of checking the development of micrococci in culture solutions, especially acetic acid, but the mineral acids, also carbolic acid, it is said, have no such action.

The *bichloride of mercury* also possesses this quality to a very marked degree.

Now let me, for a moment, review this subject in the light of treatment, which to us is certainly of greatest importance. We may look at present upon the *micrococcus* as associated with the malignant symptoms of all complications known as "blood-poisoning." It is found in erysipelas, in puerperal septicæmia, in diphtheria, and in malignant measles. Experience has already taught us that alcohol, the vegetable acids, calomel, or corrosive sublimate, are the drugs *per se* in septicæmia.

The action of alcohol and calomel is too well authenticated in puerperal septicæmia to doubt their efficacy.

We know of late how surprising a result will often attend the use of alcohol and *corrosive sublimate* in malignant diphtheria, and also the value of vegetable acids, especially lemon-juice and claret, in this dreaded disease.

My cases simply illustrate one part of the subject. In this recital I do not allude to the other death-producing complications which are so universal. Children with measles will die of cerebral complications of pneumonia, of enteritis, and entero-colitis—with these we have nothing to do at present. Their treatment will, of course, depend upon the lesions; quinine, opium, hot baths, poultices will all take part.

I have simply brought forward the subject of "blood-poisoning" for your consideration, and as these remarks are based upon the careful study of only one epidemic, they cannot be submitted as conclusive, but simply as illustrative of what may, at some future time, be accomplished by studying, not merely the bacteria anatomically and physiologically, but by experimentation with bactericides as antidotal in their action in diseases they may cause or complicate.

The conclusions which seem warranted by the statements of this paper, and by observations made in other cases in the Hospital, are as follows:—

The *micrococcus* is found in the contents of pustules and vesicles, and also in the blood taken from the measles-papule in ordinarily mild cases, without its being present in the blood taken from the punctured finger. In severe cases, called malignant in this paper, owing to the rapid appearance of morbid

symptoms, the blood shows early in the attack numerous patches of micrococcus in the field.

In cases of rapid sthenic disease with high temperature and great tissue change, the evidences of large quantities of fibrine with a tendency to coagulation are manifest. The rapid production of micrococci soon gives the mechanical impediment, and if stasis takes place from any other obstruction to the circulation, clots rapidly form.

The non-appearance of clots in malignant fevers attended with fluid blood, such as low forms of typhus, diphtheria, etc., is simply due to the fact that rapid tissue changes have resulted in decomposition, instead of into fibrine-forming substances—no fibrine is formed, hence no clots—but the micrococci are present all the same. These cases are held by some to be the malignant ones, but I think the *foudroyante* character of the others, just mentioned, entitles them to be placed in the same category.

But the micrococcus, if left unheeded, may attack the white corpuscle as distinctly seen under the microscope, and destroy its contents. The red cells also change in appearance, and finally probably become, to all intents and purposes, useless in the economy. When such a condition is seen by the microscope and found extensive, a fatal prognosis can be given, despite the most active treatment.

In cases where the white blood cells are as yet unaffected, treatment, when active, will be followed by good results, provided the other complications, as visceral inflammation, etc., are not in themselves excessive.

Alcohol (whiskey in our cases) seems in some way,

when given in large amounts, to check the progress of the marauders, to arrest the process of destruction, and, if needful, can be associated with quinine and iron in small repeated doses, digitalis perhaps, and frictions, baths and poultices, etc. As we have seen, the symptoms presented are contemporary with the changes going on within the blood; they may, *in lieu* of a careful microscopic examination of the blood, be taken as a gauge for treatment; knowing what can and will take place, early active treatment will give the patient some chance for the future.

A CASE

OF

CERVICAL LYMPHADENOMA, TREATED BY THE APPLICATION OF EARTH.

By

ADDINELL HEWSON, A.M., M.D.

[Read, September 6, 1882.]

I AM induced to present to the College this evening a report of a case, the history of which is singularly complete up to date as to its cause or origin, and its steady development up to the time of my taking charge of it as well as to its symptoms and their rationale.

Indeed my reason specially for wishing to present the patient to-night is not only to describe the symptoms of this interesting disease but to show that it has been more than arrested in its course—that it is actually undergoing positive dissipation, and, that too, by what has alone been used with it from the very first of my treatment.

The following is the history :—

P. M., aged 53 years, was born in Ireland, of healthy parents, who brought him to this country when he was over 17 years of age. His father died not long since, aged 72 years. His mother is still alive, but has always been, since her earliest recollec-

tions, a sufferer with arthritic rheumatism. There is no tendency on either side of his family to phthisis. He had always, prior to the first manifestations of his disease, enjoyed most excellent health, and by a long practical experience in machine shops, foundries, and in the working of steam engines, he was well qualified to fill the post of an engineer, which he obtained at Girard College in the year 1877, and has faithfully filled ever since. There, as I have been informed, he has displayed zeal and industry not only in his own duties, properly speaking, but by making himself always useful whenever occasion might occur. Thus on the occasion of the preparations of the meals of the college he was not only watching the conditions of the engines and boilers of its grand culinary arrangements, but was always willing and ready to assist any one with whom he might there be brought in contact. So on a summons he was prompt to go from one of the very hot rooms of the kitchen to one of an equally opposite temperature, in the refrigerator, large rooms made as cold as ice could make them in which meats and other articles of food were perfectly preserved.

It was after one of these exposures that he was seized with the first symptoms of cervical lymphadenoma. Then two weeks before Christmas, 1880, one year and nine months ago, he was long enough in one of these refrigerators to get all his clothing thoroughly wet and his body so chilled that he became prostrated. This exposure was followed by a general reaction and a pain in the right supraclavicular fossa. These chills and fevers were constantly recurring for several days, and those of

the second period of twenty-four hours were specially characterized by a kindred pain in the left supra-clavicular fossa. The pains of both sides subsequently extended up to the occiput and from there down the spinal column, becoming more intense at its fundamental end. With the latter there was numbness or partial want of sensation not only of his lower extremities, but all over except, as he says, in his hands. His throat was from the first sore and his speech about a year ago became so indistinct as not to be readily understood. Three months later his hearing, on the left side, was absent, and five months later he became partially deaf in the right, not being able to hear then the ticking of a watch beyond twelve inches.

The temperature of the neck was at first highest on the left side, and continued so until last Christmas when it became, by far, the highest on the right side. He had then been treated from the first for sore throat, by gargles and medicines, anodynes, alteratives, and the like. These he continued up to the time of his coming to me. During the period to which I have referred he had both of his ears inflated by the Eustachian catheter and gum bag, by an attendant at the clinic of one of our public charities. This inflation was followed by much pain, especially in the left ear, which continued up to the time of my first seeing him, when hearing in that ear was absent. I then tested the hearing of the right ear by an English lever watch and so showed it to be no more than eight inches.

When I first saw him, June 21st, 1882, he presented a most singular appearance with his occiput

and chin both drawn downwards and forwards, and his shoulders upwards, inwards, and forwards. His face was the very picture of distress. The room in which he was had, in the little time he had to await my coming there, become so impregnated by the smell from him as to make it stink like an opossum's kennel. This was still perceptible there more than an hour after his leaving the room. The patient has always admitted that this fetor came constantly from him and was often the subject of comment or of complaint by those about him. A close inspection at the first soon satisfied me that it was from the skin of the sides of the neck and from there only that the whole stench came. This was demonstrated as a fact beyond all cavil or doubt, by its complete dissipation in a few days by the dressings I used.

The poor fellow was sitting astride of a chair in the room, when I went in, gasping for breath, with a loud, irregular, stridulous respiration, whilst he supported his head by both his hands. By considerable effort he made me understand that his distresses all came from the tumors on his neck which he had all ready to uncover and show to me. Whilst he was doing so, however, I had walked partially around him, and got my first full view of his neck from behind. It there looked as if he had two necks attached to the base of his head. The growths on the right side, projecting beyond the line of the outer border of the trapezius there for a width equal to that of the whole neck and the excrescences on the left side, produced this appearance. He then managed to tell me that his sufferings were incessant, that he had not been able, for a long time, to maintain his head when erect

without the aid of a hand or some other means of support except for a few moments; and that he could not, from a very early date of his troubles, lie down and sleep in any position over a period of twenty minutes, as he was always then startled by a feeling of impending suffocation.

Seeing that this case would require some lengthy and careful investigations before I could properly prescribe for it, I directed the patient to call on me the next day at my office during my hours there, to bring with him samples of his urine voided at bedtime and on rising in the morning, and not to use anodynes on going to bed that night.

He was with me early the next morning, and not only repeated the details which I have given, but gave me full opportunities of discovering and studying all his symptoms.

The urine he had brought possessed a sp. gr. for that of the evening of 1017, and for that of the morning of 1021. Microscopic examination revealed the presence of phosphates. The quantity of urine voided in twenty-four hours was, according to his statement, about normal.

His pulse was decidedly frequent (102) and feeble, evidently somewhat so disturbed then by nervous agitation. His respirations were likewise disturbed, and the impulse of his heart was feeble and diffused.

The mucous membranes of his lips, mouth, and tongue were very red and swollen. This condition being most marked around the gums and being associated with positive looseness of the teeth, at once suggested the probability of ptyalism being present.

The patient stated that he could not masticate any solid food or swallow any hot liquid.

The tonsils and uvula, as also the half arches and walls of the pharynx, were likewise much swollen and reddened, and efforts to inflate the middle ears by forced inspirations and expirations on his part whilst the mouth and nostrils were held tightly closed, showed that the internal meatuses were both completely obstructed, and although he was then free from all signs of catarrh in either antra and forehead, it was evident that the obstructions in the internal meatuses were, like those of the external, due to the immense size of the tumor on the outside of the neck.

The contour of these tumefactions was limited essentially to the lateral regions of the neck. Starting in the supra-clavicular fossa on either side they were greatest at first, posteriorly, along the planes of the trapezii muscles, as can be seen on the diagram or tracing which I made before beginning the treatment (see Fig. 1). Those muscles were, on both sides, much stretched and pressed backwards, and were very markedly defined by this backward prominence of the tumor.

The tumor, then, on both sides extended high up behind the ear, covering both mastoid prominences, but did not extend further back there than the outer attachments of the trapezii to the occiput. In front of the ears they went up to the zygomatic processes of the malar bones projecting outwards in front of each ear to more than an inch from the jaw.

The front lines of these projections dipped down to the base of the jaw on either side for an inch in front of the anterior auricular processes of the ex-

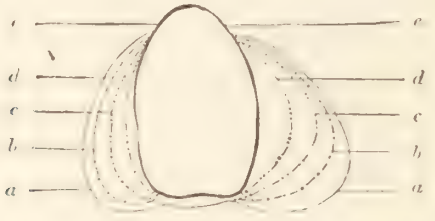
ternal meatuses, covering up to some extent the ears, as seen from in front, and giving a strange appearance to the face by hiding the ears, and this was intensified by the hair of the beard there.

The swellings extended forwards on both sides of the face along and close to the base of the lower jaw, and as far forwards as the front of the submaxillary glands. These glands themselves were greatly increased in size; that of the right side being of the size of a hen's egg, whilst that of the left was half that size. They both projected for more than half their thickness beyond the line of the lower jaw, and furnished by the anterior extremities the limits of the growth there. Indeed a line drawn perpendicularly downwards from those extremities showed that the tumors of both sides did not come any further forward, lower down than those points, and a critical examination of the region of the larynx and of the sternal extremities of the sterno-cleido-mastoid muscles, showed that the central portions of those muscles passed through the tumors, or more accurately, that the tumors were developed there on the *inside* as well as the outside of those muscles.

To preserve as definite an idea as possible of these growths, I took and kept one set of tracings by means of a long strip of sheet-lead one inch wide and one-eighth of an inch thick, which I placed, when well warmed by friction on their surfaces, along lines extending from the outer margins of the trapezii forwards and two fingers' breadth below and parallel with the base of the jaw, and as far forwards there as the anterior border of the sterno-cleido-mastoid muscles. These secured the *three fixed points* by which I

could subsequently make tracings for comparison. These transverse tracings which of the first day are represented by *a*, Fig. 1, I carried back to the median

Fig. 1.



line of the spine and forward to the pomum Adami, so as to complete an oblique circuit there. The details of these tracings I subsequently confirmed the accuracy of by the free use of a pair of calipers and of a tape measure. And further, as the most prominent points of the tumors were on each side in the planes of the trapezii, I made another set of tracings (see Fig. 2) representing their vertical outlines in that plane. This I did by means of a long pencil whilst the patient's body was entirely uncovered, and he was lying with his back, neck, and occiput on a surface to which I had secured a large sheet of paper.

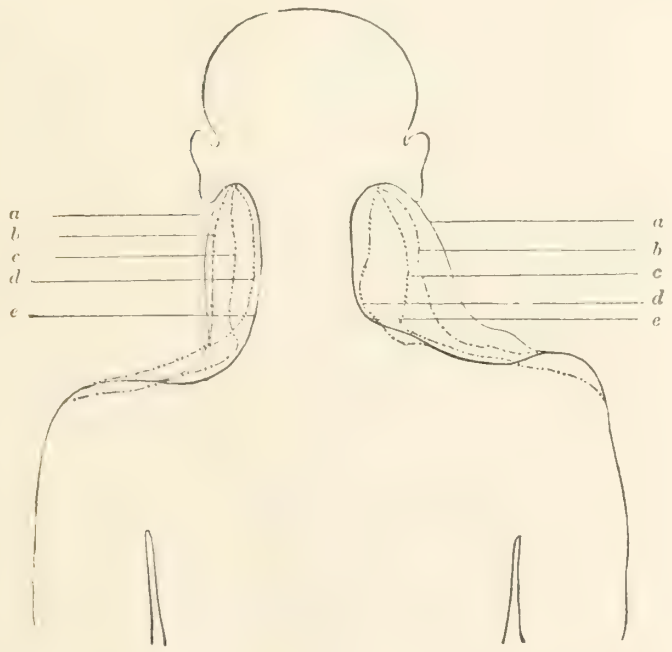
The outlines of the trapezii, as well as those of the back of the head, of the ears, and of the shoulders, which were essential to complete this set of tracings, I made by a continuous line.

In Fig. 1, I have also given by the innermost line, *e*, a tracing which I got from the left side at the end of the third week after my beginning treatment in the case, and when all the tumefaction had so disappeared on that side that this line might be fairly accepted as

representing the natural line of the contour of the neck. To secure a corresponding line for the right side of Fig. 1, I doubled the sheet of paper on itself along the line, which would represent the middle of the pharynx, larynx, and spine, and made a tracing for it, like that of the left.

By comparing these two sets of continuous lines in the two figures, one can readily see what were not

Fig. 2.



only the differences in the growths of the two sides, but what were their actual thickness and relative forms on the day I began treatment.

The actual measurements which I made before my first dressing were by a line drawn across the back of the neck, just three inches below the occipital protuberance, seven and one-half inches—a like line in the back, horizontal with that of the clavicles in front, eleven inches, and a line straight across from the tip of one shoulder to that of the other, fifteen and one-half inches.

The measurements obtained after one week's treatment were, for the first of these three lines, *b*, six inches, a reduction of one and a half inches; for the second line, *c*, nine inches, a reduction of two inches; and for the third line, *d*, a trifle over sixteen inches, which being a difference in singular contrast with those of the other two lines, caused momentary surprise, but was soon found to be explicable by the increased freedom of motion of the shoulder of the right side, which allowed it to be carried well back.

At the end of the second week similar measurements, indicated by the line *e*, the third set across the backs of the neck and shoulders, were respectively five, nine, and sixteen inches, showing a change only for the first of the series.

Then, as I have said, the original infiltration into the subcutaneous cellular tissue had become so completely dissipated that the real *lymphomatous* character of the growth was to be readily recognized, and was commented upon by Prof. Jas. Tyson, who saw the patient on the morning of the fifteenth day, when I was applying the dressings. A sulcus had then formed on each side, separating very well, and defining clearly the cervical from the supra-clavicular portions, as can be seen at *c*, in Fig. 2. These sulci

became very great during this, the third week, especially on the right side, and they grew essentially at the expense of the truly cervical portions of the tumor.

At the end of the third week of my treatment the posterior borders of the tumors on each side had so ceased to be on the plane of the trapezii that I could not make any more tracings on Fig. 2, to be used for correct contrasts. On the contrary, as can be seen in Fig. 1, the widest or most projecting portions of the tumors were then in the centres of the sides of the neck, that of the left side being nothing more than what would represent the natural contour of the neck there. I therefore ceased to make tracings on either figure.

The patient had now not only recovered freedom of motion, lateral rotation, and flexion of his neck, but could hold himself perfectly erect. This has made an actual difference in his stature of one inch and a half over what it was at his first visit to me. His breathing became natural as early as the fifth day of my taking charge of him. He was then (June 26) able to lie down and sleep without any anodynes for twelve hours at a time, a thing he had not been able to do since the first of his attack, although he had always been treated by the free use of vegetable narcotics, bromides, and other saline sedatives. I had forbidden his using anything of the kind on the night of my first seeing him. With this came improvement of his pulse, his appetite, and his general nutrition. These improvements have continued steadily going on up to the present time, the end of the eleventh week to-night since I first saw him. His complexion, which was then markedly leukæmic, is now, as you

can all see, ruddy and as healthy-looking as could be desired. He has regained his hearing perfectly on both sides. He has no signs of pharyngeal irritation or constriction. His voice is full and clear. His pains and hyperæsthesia of the integuments covering the growth disappeared in the first week, and have never returned.

Treatment.—This patient had never been without treatment since the onset of his attack. He saw his family physician on the next morning, who treated him first for a bad cold; then, and up to a short time before I saw him, by various constitutional remedies, such as iodides and bromides and other alteratives, and locally by the application of tincture of iodine, blisters, and poultices. With them all the patient had, however, not obtained the least sign of relief. On the contrary, he said he had continued to grow worse up to the time of my prescribing for him. I then advised him to stop the administration of these remedies, and fearing that so sudden and complete a stoppage of the anodynes to which he had long since become habituated, might occasion distress to his nervous system, prescribed the tincture of avena sativa, ten drops of which he was to take in hot water at bedtime every night. He took but two doses of that remedy, for on the third night he was so freed of all pain and distress that he went to bed without taking any, and slept the night through. With this single exception I have not given him anything since his first visit to my office, but have relied solely on a daily application of yellow clayey earth. This I have put on in the form of a smooth paste, thick enough to make it hold fast to the integuments of all parts

where there was any swelling. This paste I made with water by means of an ivory spatula. By the latter I also made the application all over the tumefaction to the thickness of an eighth of an inch, laying on top of this a firm film of cotton batting, so as to have the clay fixed and held tightly in its place after it became dry, and until the shrinkage of the tumefaction would become such that free spaces were readily to be detected between the inner surface of the dry clay and the outer surface of the tumor. The necessity for renewing the clayey dressing was apparent every day. Indeed, after the patient had recovered his power of free motion of his neck, he did not maintain the applications in their places a whole day at a time. He never, however, washed the parts. What was left of the clay hanging to the integuments showed in itself the fact of shrinkage following the drying of each application. I dressed him in this way with the clay only once every day for eight weeks, and that in the evening, so that he did not lose any time from his work, about which he was anxious. His anxiety not to lose any time from his work prevented me from getting any photograph, which I am in the habit of doing when I undertake such cases.

At the end of two months the shrinkage was such that the tumors on both sides were no longer so easily disturbed as regards their coverings by the movements of the neck. The tumors themselves were then so reduced in thickness that they were, on the left side, only small subcutaneous fragments or nodules of what was originally there, and on the right they occupied small spaces in the supra-clavicular fossa,

the outer surface of the sterno-cleido-mastoid and the like surface of the submaxillary gland, all being so free by passive motion that their coverings with the earth were nothing like as easily disturbed as they were originally by the movements of the neck. For these reasons I entrusted the daily applications to the man's wife and himself. He says, however, that the applications do not hold as long and as well as my most recent ones.

In spite of all obstacles to the purely earth treatment, I am well satisfied with the progress of destruction which has taken place in this case, and my special object of bringing the patient here this evening is to show his present condition, in order that it may be compared with that which the subsequent treatment will effect.

THE BACILLUS TUBERCULOSIS.

By

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[Read, by invitation, September 18, 1882.]

Our definite knowledge regarding tuberculosis dates from the day when experiments were first made to prove the specificness of the disease. We all remember well what a kind of thrill went round the medical world when Villemin, in 1865, first announced that he could inoculate the disease by means of tuberculous virus. We recall, too, the numbers of experiments which were performed at once with conclusions, some in confirmation, some in refutation, of his views. Chief among the opponents of them at this time was Cohnheim, who, with many others, stoutly maintained that he could produce the disease by the inoculation of other kinds of matter, even with wholly innocuous and inorganic matters, like elder pith, India rubber, etc.; in short, with anything that would produce irritation and caseation, which latter process came to be looked upon as a *sine quâ non*. Hereupon followed the doctrine of caseous pneumonia, a process secondary to catarrhal or croupous pneumonia in a vulnerable subject, and the sequence was catarrhal pneumonia, caseous pneumonia, phthisis. But the

pathologists soon saw that neither catarrhal nor croupous pneumonia was wont to result in caseous pneumonia, and that, while phthisis was a disease so common as to carry off one-third of the productive population, neither of the so-considered causative affections occurred in sufficient frequency. Buhl then described a desquamative pneumonia, a form different from any other, whose business it was, as it were, to result in caseous degeneration. The pathology of tuberculosis now became involved in the hazy mists of clinical speculation.

The next direct observations were made in the experiments of Tappeiner, who saw that dogs, which naturally enjoy great immunity, became tuberculous when forced to breathe inclosed in boxes containing atomized tuberculous matter. These simple and striking experiments were variously repeated again with contradictory results. But the idea of specificness was thus launched again, and very soon thereafter when the oculists began to produce the disease by the inoculation of tuberculous virus in an organ where the whole process of development could be seen by reason of the translucency of its structure, the bulk of evidence was in favor of regarding tuberculosis as a specific disease. Cohnheim and Salomonsen now published the results of their inoculation experiments upon various animals, arriving at the conclusion that tuberculosis could be produced in no other way than by the introduction of tuberculous matter. Not the presence of giant-cells, the formula ran, not the process of caseation characteristic of disease, but solely the capability of inoculation. Thus there was gradually evolved the fact of the

specificness of the disease, so that by the beginning of this year the doctrine had deepened into a general conviction.

Then so soon as this prime factor was acknowledged, the idea was naturally awakened, or rather re-awakened, of the presence of a *contagium vivum*. Klebs, who is always first in this field, in point of time at least, described the actively-moving *monas tuberculosum* (bacterium). Schüller and Toussaint followed with micrococci, and Aufrecht followed them with two forms, one for scrofula, one for phthisis, one a micrococcus and one a bacillus. There was no satisfaction in these discoveries, which rested simply upon microscopic appearances, and they soon took their place silently among the list of the lost. By the beginning of this year Cohnheim was still able to say, "The principle of the infection in tubercular affections still remains a problem unsolved."

How Koch solved this problem is a matter of history already in the short time which has elapsed between April 10th, the date of his first publication, and the present day. He had already made fame in an obscure country town with his researches upon surgical infection, erysipelas, and milzbrand. But his reputation was chiefly confined to his co-workers, whose attention soon became fixed upon him on account of the accuracy of his observations and the precision of his statements. The recurrence of a particular form of bacillus under the same conditions of investigation concerning tuberculosis soon convinced him that he had something more to deal with than a mere accident or coincidence. The same means were at hand

which had been adopted with such success in isolating the bacilli of milzbrand, and the principle of the solid culture soil was modified to secure the development of the bacillus tuberculosis. In this way he soon arrived at the conclusion that he had to deal with a special form of parasite, and the question only was as to its exact relationship to the disease. But first let us speak of the anatomy or morphology of the new discovery.

The bacillus tuberculosis is rod-shaped, hence its name, varies in length from $\frac{1}{1200}$ to $\frac{1}{3500}$ inch, is *absolutely motionless*, produces spores, 2–4, in the body, and is blunt at both ends. It is present in greatest abundance where the disease is in most rapid progress, and may be absent where the disease has ceased to spread. It is found in the interior of the so-called giant-cells, which are in reality but capsules for it, like the capsules of trichina.

But no one, certainly not Koch, would claim to differentiate a bacillus by its gross physical appearance. Milzbrand is easily distinguished from tuberculosis by the fact that its bacilli are pointed at each end, while the bacillus tuberculosis is rounded or blunt; but such differences are of value only when both bacilli are present for comparison. The spirochætæ of relapsing fever are distinguished at a glance, because they are spiral and not straight—because they are, in fact, not bacilli (rods) at all. The bacilli of mouse septicæmia are distinguished at a glance because of their extreme minuteness, and the bacilli of rabbit septicæmia are distinguished, because they are more like micrococci than bacilli, that is, they are more elliptic than linear. But these distinctions are of

value only in the mycological laboratory, where they all occur with equal frequency, and would not interest us as clinical practitioners. So far as concerns morphology alone, the various bacilli are at the first glance very much alike, often so much alike as to be indistinguishable; but in this respect they are not unlike many other microscopic structures, say the spermatozoids, which are apparently very much alike and yet are very different.

But if the bacilli do not differ much to coarse inspection, so to speak, do not differ much in their anatomy, they do differ widely in their chemistry, which brings us to speak at once of the color tests; for the action of the coloring matters upon the bacilli is an expression of their chemical affinities. And here we find very great differences among the different bacilli. Pretty much all the micrococci can be tinted of any color indiscriminately, blue or red or brown, etc., but the bacilli show marked preferences for, or perhaps only become visible with, the use of certain hues. In the case of the bacillus of tuberculosis, for instance, the specimen supposed to contain it must be first tinted, then decolorized, and tinted again, to bring it out with definiteness and distinctness. The bacilli of lepra, which closely resemble those of tuberculosis, can be colored only when fresh, not after desiccation; but, hardened in alcohol, they may be colored for years with gentian-violet or fuchsin. The spirochætæ of relapsing fever can be colored properly only in blood, but not in the tissues, with fuchsin, methyl-violet, or gentian. In section they can be colored only with aniline-brown.

I may best serve the purpose of my invitation here,

I think, if I confine myself as closely as possible to the text presented, the bacillus tuberculosis, and I construe the occasion to mean a demonstration rather than a declamation. I have therefore had drawn merely as a means of coarse comparison the various sketches of micro-organisms of which I have spoken, and which I now exhibit, and, having shown them, proceed to the clinical demonstration of the bacillus tuberculosis in the sputum.

This specimen of sputum, which answers to the old classical description *sputum rotundum fundum petendum*, I picked up in the hospital, or rather it was kindly given to me by an interne, Dr. Devlin, as taken from the cup of an advanced case. I have already prepared a specimen from it which I have here, and I proceed to repeat every step of the process necessary to its exhibition before you, commencing with the raw material. I select a small fragment of the sputum, and spread it with a knife as thin as possible over pretty much the whole surface of a cover-glass. The next step is to fix it, for, if we should accidentally omit to fix it, we should have the albumen precipitated in forms which would puzzle the most profound mycologist. We may fix it, if we please, with glycerine-brown, a solution of aniline-brown in glycerine, and for photographic purposes this is the best procedure, but for ordinary quick demonstration we fix it best by heat. I pass it, therefore, two or three times through the Bunsen's jet, specimen side up, of course, taking care not to hold it in the flame lest it be burned up. The specimen is now fixed; it is dry, as any one may see, but it is not burned.

I have here now upon the table all the crude materials for the manufacture of the color-tests in common use. First is the bottle of aniline oil, a dark amber-colored fluid, and next is methyl-violet, which we select because it gives us the quickest results. The aniline oil, mixed with distilled water, and allowed to stand four or five hours at least, with occasional agitation, gives us this soapy-looking fluid, which is labelled "the aqueous solution of aniline oil." The methyl-violet powder dissolved in alcohol gives us this concentrated alcoholic solution of methyl-violet, and these two solutions mixed, in the proportion of one hundred grammes of the first to eleven grammes of the second, give us this deep violet, in thin layers iridescent, fluid which constitutes what we might call the first color. Several watch-glasses having been half filled with the first color, we let fall upon the surface of each, taking care that each rests upon and does not sink below the surface, a cover-glass with the specimen of sputum. We select several specimens because we might fail to find the bacillus in any one. Here now the specimens rest for half an hour, while we take up another portion of our subject.

Let us suppose that we will find in our preparation some specimens of the bacillus tuberculosis, or at least take it for granted that we can find it in most specimens of phthisical sputum, or in sections of tubercular structure; and on this subject there is now no doubt; the question at once arises as to the significance of the bacillus. The bacillus need not necessarily be the cause of the disease, any more than any one of the numerous micro-organisms found in the stools

need be the cause of typhoid fever. In other words, presence does not imply pathogenesis. Koch might have felt convinced that he had found in this bacillus the cause of tuberculosis, but he would not be able to convince others to that effect unless he could produce tuberculosis with the bacillus. And that is what he did. But he had at first to isolate the bacillus from everything else. Koch had already effected this isolation of individual germs in other diseases, notably in milzbrand and forms of septicæmia, and he at once undertook to grow the bacillus upon the same soils. He soon found, however, that the bacillus would not grow, or would not grow well enough, upon any of these culture substances, and then he commenced to prepare for it its own native soil, the soil to which it is indigenous, so to speak, viz., blood. One might almost consider this thought as an inspiration of genius, if it were not so strictly commonsense. The problem was, then, not to get a solid substance out of blood, for any one could do that in the process of coagulation, but to get a substance that would keep and would be clear. Sterilization and solidification at the same time were the factors sought. With the patience of science, he worked this problem out. Having drawn fresh blood into a perfectly clean vessel, he allowed it to stand absolutely at rest for twelve hours, when he withdrew into glass pipettes, previously sterilized by heat, the supernatant serum, and let it fall into test-tubes likewise purified and closed with plugs of sterilized cotton. These tubes were now subjected to a temperature of 58° C., one hour a day for six days, when full sterilization is effected, as proved by the fact that

the blood will keep for months or years if the plug be not removed. Then solidification is effected by raising the temperature to 65° C., always in a water-bath, of course, withdrawing the tube from time to time to observe the period of gelatinization. Thus prepared, the tubes are inclined sideways somewhat, to offer a larger surface simply, and the bacilli, best from a tuberculous deposit in an animal artificially inoculated with the disease, are inserted upon the surface of the prepared blood. In other words, the seed is sown. For a week nothing is observed. At the end of ten days, however, a scaly appearance begins to show itself, the scale as it grows lifting itself from the subjacent soil and curling up upon itself like an old epidermis cell. It is very friable, and is very easily detached, in which regard it differs widely from other micro-organisms, from milzbrand, for instance, which goes down into the substance of the soil as a film or cloud, and gradually invades the whole mass. This scale upon the surface of the blood is the tuberculosis itself. A fragment of it put under the microscope is seen to be composed wholly of bacilli. It may be dissolved in fresh glycerine and injected, or it may be inserted with a needle or platinum wire, and thus inoculation is effected. The wound heals in a day, leaving no trace, but in about two weeks the nearest lymphatic gland enlarges; in a few days more other glands are affected, and the animal begins to emaciate; in short, to present the general signs of tuberculosis. Now it may be allowed to die of the disease, or it may be killed at any time and examined, when the organs present the picture of tuberculosis in de-

gree corresponding to the amount injected. All these things have been so faithfully rendered before that I do not dwell upon them, save to remark that while some animals, notably the dog and the rat, enjoy comparative immunity against spontaneous tuberculosis, there is no immunity to the artificial disease.

But this very fact of immunity and liability is an argument for the specificness of the disease. Thus the minute bacilli of mouse septicæmia kill house-mice but not field-mice. The bacilli of milzbrand kill mice, but not rats. The bacteria of rabbit septicæmia are always quickly fatal to rabbits and mice, and may be communicated to sparrows and doves, but do not affect guinea-pigs and rats. Age plays the *rôle* in these experiments, for which we are prepared by clinical observations concerning the acute infections. Thus milzbrand will affect young dogs and rats, but not older animals. Perhaps the most singular action in this connection is observed in mouse septicæmia, the bacilli of which kill young rabbits, but produce only a local affection or inflammation, a kind of erysipelas, in old rabbits.

We proceed now with our experiment. We observe, as we lift the cover-glass from its methylene bath, that the preparation is deeply stained. We shake off the superfluous color and immerse the specimen in the decolorizing solution, which consists of nitric acid (German Pharmacopœia) one part to distilled water two parts, by volume. Moved about in this fluid, the color quickly disappears. Held between the eye and the light, the specimen looks almost colorless; but in washing off the superfluous acid,

under a small stream of distilled water, it is seen that a faint tinge of color returns. The specimen is now ready for the second color, the concentrated aqueous solution of vesuvin, which colors all the field brown. One drop of the recently filtered, but still quite thick, solution is let fall upon the cover-glass while it is tipped about to diffuse it over the whole field, the cover-glass is pressed upon the slide, and the examination for bacilli commences. As we have already a preparation showing them in this specimen, we need waste no time in looking for them now.

Having said something of the color and the culture, let us pass now to the subject of control. No one would expect to cure a case of any disease by a mere knowledge of its cause. This is of course the prime factor, and is alone worth all the rest. But our knowledge of the bacillus tuberculosis is still too new for any consideration of agents destructive to the parasite. Nor is it probable that any such agent will be discovered, or will be ultimately necessary. We do not know any agent destructive to the trichina spiralis in the body, and yet the discovery of the trichina has almost put an end to the disease. That is, it has limited it to the ignorant and the careless. He who studies disease from the standpoint of control has a much wider range of intellectual vision than he who looks upon it simply from the standpoint of the common idea of a cure. But the bacillus tuberculosis from the standpoint of control must first be universally acknowledged.

It comes to us then with some points characteristic of it in its morphology, or we might say its anatomy; secondly, it has more definite chemical properties, as

shown in its behavior to the analine dyes; thirdly, it grows only upon a definite soil, different from many other bacilli, hence it has, we might say, speaking of it as of any other vegetable, its own agricultural properties; fourthly, it grows in its own peculiar way; and lastly, supremely important in a differential point of view, it has its own specific, exclusive, physiological properties, as evidenced by the constancy of its action upon pretty much the whole animal kingdom. Is there any other factor in all etiology that comes with stronger credentials? Let him explain it who can, however, the singular psychologic fact remains that every one is willing to admit all things in the case of milzbrand, but not in the case of tuberculosis. It is, perhaps, because milzbrand so seldom affects mankind.

Koch never rushed into print with his discovery. He studied the bacillus tuberculosis thoroughly before he published the existence of it, and he determined the parasitic nature of it in the very simplest way: by the observation, namely, that it lives only at a temperature between 30° and 40° C. Milzbrand, its congener in so many ways, lives and produces spores in the coldest countries, and we see the worst epidemics of it in Siberia and other lands of the north. But milzbrand is not a parasite in the true sense of the term. The bacillus of milzbrand gets into the body of animals only accidentally, makes a little excursion, the Germans say, when it leaves the field, to be swallowed into the body of a cow. But the bacillus of tuberculosis requires body heat to live and thrive in, and produces no spores even two degrees above or below its natural climate.

So the bacillus tuberculosis is a true parasite, and, when we recognize this fact, we almost get possession of it. It is like tracing a wild beast to its lair. The bacillus tuberculosis is not wild. It is motionless, sluggish mostly in its growth, but it is all the more cruel on account of these facts. Desiccation does not kill it, if it be kept sufficiently warm. Koch inoculated guinea-pigs with parasites from desiccated sputum a month old, and nevertheless produced the disease. One of the best of our own mycologists put it well when he said that "the mud of the gutter to-day is the dust of the air to-morrow." It is the sputum which disseminates the disease. In spontaneous tuberculosis the disease always begins in the lungs. Even in cases of basilar meningitis, a careful search will turn out nearly always a caseous bronchial gland as the initial lesion or depot whence is the local or general invasion of the body.

So here we have the clue to the control of the disease, and, when the phthisical patient pulls out his sputum-saturated handkerchief, he opens the box of Pandora on the public highways. If I had to make practical response to the query what is the means of absolute control, I would say, in the light of existing knowledge, fire, and fire alone. No kind of bacillus will live in the fire. But a patient with phthisis cannot sit and spit in the fire. In the laboratory at Berlin there are many poisons floating in the air. The air of one of the rooms once became so permeated with milzbrand bacilli that a mouse, which is exquisitely sensitive to it, could not live in it more than twenty-four to forty-eight hours. Yet all the tools and instruments are thoroughly disinfected, that is,

freed from germs, by the hot air- or water-bath; or, better still, when adoptable, by the Bunsen's jet. But the glass-slides and cover-glasses could not be treated in this way. So there stands upon the table in each room a large glass vessel holding half a gallon at least, and that much concentrated sulphuric acid is poured into each. Into this vat is thrown every slide and cover and test-tube that has been used, and once a month it is all cleaned out. Perhaps phthisical patients who may not be isolated as yet, as are other cases of a less malignant malady, might be restrained from disseminating the disease by the use of similar smaller portable vessels. But, however it be accomplished, the prime point now in prophylaxis is the recognition of the fact that it is the sputum which chiefly conveys the disease by contaminating our common reservoir of air. Just now it occurs to me, under the inspiration of this occasion, what many a classical scholar no doubt has thought of here without such stimulus: the disgrace it used to be held to be in the days of ancient Rome for a man to expectorate in public. And with our knowledge of the danger which lurks in the sputum often, how much graver insult it is than a mere breach of propriety, how much deeper offence than a mere disgrace.

CLINICAL OBSERVATIONS ON ALBUMINURIA, BASED UPON A STUDY OF SIXTY-TWO CASES SEEN IN PRIVATE PRACTICE.

By

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[Read October 4, 1882.]

During the last eight or nine years it has been my habit to keep a record of the examinations of urine I have made. That record now contains notes of between sixteen and seventeen hundred examinations, and a study of it recalls to my mind, with more or less distinctness, the histories of sixty-two persons suffering with albuminuria.[1] In a number of instances I have been able to follow cases for a series of years; from this circumstance in particular, the number of years the patients were under observation, I have been enabled to satisfy myself of some clinical facts, particularly in reference to the diagnosis and prognosis in this complaint, which I hope may be worthy of your attention.

That which has most impressed me is the impossibility of making a prognosis, with any degree of exactitude, in most cases of Bright's disease. Of

[1] Many of these cases occurred in the practice of my father, Dr. J. Forsyth Meigs, but a large majority of them I saw from time to time myself.

course, in the plainer ones, the decision is easy; if asked an opinion in a case with a large, or even moderate amount of albumen in the urine, with increasing heart failure, and evident decline of strength and vitality, with headaches and the peculiar white complexion of the disease, it is easy to prophecy that such a person will not live long, and the prophecy, nine times in ten, comes true; if called, however, to decide questions of the future for a young man of thirty-two or three, previously well, but for some weeks complaining of headaches, malaise, and boils, and then examination of the urine shows a slight amount of albumen, granular and hyaline casts, and abundance of rather small oxalates, the question of the future is not so easily decided. I have seen such cases go on pretty well for a few months, then suddenly have convulsions, and die in a few days. Again, persons presenting identical symptoms, after being sick a few weeks or months, entirely recover. To my mind it is impossible, in the present state of our knowledge, to anticipate the future, and the patient should always have the benefit of the doubt, and be shown the brighter rather than the darker side of the picture.

I know of three persons in whose urine I found albumen and tube-casts, accompanied with all the other signs of Bright's disease more than eight years ago, who are yet alive. One is a lawyer in easy circumstances, who was taken sick in January, 1874, when he was fifty-six years of age, with what appeared to be a bad cold, but he had extreme dyspnœa and excessive restlessness and nervousness, although he is usually very self-composed. The dyspnœa seemed greater than could be accounted for by the extent of

disease of the lungs, which amounted only to a moderate bronchitis; the urine being examined, it was found to contain a trace of albumen, some blood, and tube-casts. It should be said that this man had suffered in previous years with attacks of gravel, when he would pass blood and small calculi. Nearly four years afterwards there was still a trace of albumen in the urine, and granular casts, showing that the condition was by no means merely temporary. Ever since he is liable to severe cold, and always suffers more than common with oppression, although it usually takes the form of a common coryza. He is now sixty-four years old, and an unusually young-looking, active man of his years.

In contrast with this case, a woman of about sixty was seized in the night, having gone to bed apparently in her usual health, with an attack of oppression, and what her physician called congestion of the lungs, so violent that it was thought she would die at once; she, however, rallied by morning. An examination of the urine, made a few days afterwards, revealed the presence of a slight trace of albumen and casts. Four months afterwards there was still a trace of albumen in the urine, and granular and fatty casts, although she was up and about as usual, being merely very weak, having no apparently urgent renal symptoms. At a later examination, the trace of albumen was still found, but no casts. About seven months from the first discovery of the disease, she had another violent attack of oppression, and died in a few hours. Now what are the points to distinguish these two cases? One died after a few months' illness, and the other, having had apparently the same complaint and

almost identical symptoms, is, after eight years and a half, still alive, and more, apparently, unusually well for his time of life.

In December, 1874, a lawyer in full practice, then about fifty-four years of age, was taken with an attack of acute catarrhal pneumonia, and was also found to have albumen and tube-casts in the urine. He was sick all that winter and the following spring, the urine constantly containing albumen and granular and hyaline casts. The albumen and casts were found in the urine during 1875 and 1876, and part of 1877. On May 2d, 1877, about two and a half years from the onset of the disease, there was at last no albumen found, and the microscope revealed nothing abnormal. Since that time, although the urine was examined in 1878, 1880, 1881, and 1882, there has never been found either albumen or tube-casts, and the patient, now a man sixty-five years of age, enjoys very good health, having had no return of the disease in more than five years.

The third case is a woman, whose father, brother, and two sisters have died of the same disease. She has had a trace of albumen and hyaline and granular casts in her urine since March, 1874, when she was about thirty-one years of age. I have records of this condition in 1874, 1876, 1879, 1880, 1881, and 1882. In February last she caught cold, and seemed for some time upon the verge of a severe renal attack, having coryza, so that her nose was almost entirely stopped, and there were bronchial râles heard in the chest, but an amount of dyspnœa, seemingly out of all proportion to the extent of the disease, that could be detected by physical exploration; the albumen in

the urine also increased very much in quantity. In some weeks, however, she recovered, and this summer seems in her usual health.

The father of this patient had a severe attack of hepatic colic, and passed several gall-stones in 1877, but afterwards he continued in pretty fair health for some time. On May 9th, 1879, a trace of albumen was found in the urine; this continued to be present at succeeding examinations, but no tube-casts were found at this time, or afterwards, although they were carefully sought for, and about December, 1880, eighteen months from the original discovery of his disease, he succumbed to an attack of uræmic convulsions, being seventy years of age. A son and two daughters of this man died of the same disease some years before. The son, after having had dropsy, lived for several years in tolerable comfort, until a severe cold proved fatal. With the history of this son, and one of the daughters, I am not familiar, but the third one I saw, and she died in convulsions. The condition of this person before her final illness, which lasted only a few days, was in no way different from that of her sister, who is still living, and has had the disease for nearly nine years, and the attack which the surviving one had last March was exactly like those of which I have frequently seen other people die.

I maintain, therefore, that with our present knowledge it is impossible, in doubtful cases, to prognosticate what the result will be, and that for the patient and his family, as well as for the credit of our profession, we should be very guarded in our prognosis. The presence of albumen and tube-casts in the urine, even when found for so long a time as two years con-

tinually, is not, as shown by one of the histories I have detailed, a reason why we should necessarily give a fatal prognosis, and this is particularly the case when the patient is a person past middle life. In the face of such authorities as Beale and Grainger Stewart, many practitioners think the microscopists can better judge (by examining the urine) of the stage and form of Bright's disease they have to deal with than they themselves can. Grainger Stewart is emphatic upon the subject. On page 271 of the American edition of his work on "Bright's Disease," he says: "Some authors attach great importance to the tube-casts. They are of undoubted value in establishing the existence of Bright's disease, considered generically, but they afford comparatively little assistance in the differential diagnosis of the different forms. Any form of tube-cast may occur in any form of the disease, and at almost any stage. Indeed, all the leading varieties may occur simultaneously in one case." Beale says (Kidney Diseases, Urinary Deposits, and Calculous Disorders): "Dr. Basham thinks he can judge of the stage of Bright's disease by the character of the urinary sediment. I wish I could agree with him in this conclusion. The more carefully the matter is investigated the more convinced am I that it is unsafe in many cases to attempt to draw inferences as to the stage of the disease from the character of the urinary sediment only. Nothing but a careful consideration of every point connected with the case will enable us to arrive at a general conclusion concerning the state of the disease. . . . Dr. Basham says: 'The most untractable and hopeless form of *morbus Brightii* is that represented by

the presence of the fatty or oily cast.' This statement requires some qualification, for although it is quite true that some of these cases are terribly and rapidly fatal, it must be admitted that some live for twenty years or more, and die at last of other disorders." With regard to the presence of oily casts in the urine, and it is upon their discovery that microscopists found their gravest prognosis, Dr. George Johnson says (Lectures on Bright's Disease, New York, 1874): "The appearance of oily casts and cells excites less alarm now than it formerly did. It indicates that in certain parts of the kidney the secreting cells and inflammatory exudations are undergoing fatty transformation; but I have seen many cases of complete recovery after oily casts and cells in great numbers had appeared in the urine continuously for many weeks."

A very common symptom of renal disease, and one upon which there is not much stress laid in the majority of books upon the subject, is dyspnœa. Whenever a patient is found suffering with dyspnœa, and particularly if there is great nervousness, and loss of self-control, and anxiety, with no other fully sufficient cause for its existence, suspicion of renal disease should be aroused, and the urine examined, even if there are no other symptoms whatever, pointing toward a lesion of the kidney. These attacks of dyspnœa frequently come on with great suddenness in persons who have not considered themselves sick, and such attacks are often quite rapidly fatal. This condition has been described as renal asthma, but I believe it much more common than is usually supposed. Another symptom of Bright's disease, which

I have frequently noticed, and have never heard described, is coryza, of such a character that it gives rise to very much greater distress than any ordinary cold in the head, with this there is not much discharge from the nose, but the patient complains that he can get little or no air, except through the mouth, this being accompanied with excessive oppression, much more than the condition would seem to warrant. This symptom was first called to my attention by my father, Dr. J. Forsyth Meigs, in a case we saw together about nine years ago.

The suddenness of the onset, or perhaps it would be more correct to say, of the discovery of the existence of this disease, is often very great. In one instance, I knew of a young man, previously supposed healthy, seized with a convulsion while sitting at the supper table, after he had finished his day's labor as a wagon driver; this was followed by others, and the case ended fatally in a few days. In a second case, I was called upon by a young woman to explain a swelling of the vulva, with which she was suffering; examination of the urine revealed the existence of a large amount of albumen; this continued for some time, but after prolonged use of iron, her condition improved, and she had apparently fair health, until about two years later she became pregnant, and died within a few days after the birth of her child. In a third case, a man, fifty-four years of age, was seized in the night with epistaxis, a thing he had not had since he was a boy, examination of the urine revealed the cause, and about six weeks later, he was found one morning, insensible in the bath-tub; examination showed that he had right hemiplegia and he died in

three days; a vessel in the brain had ruptured, presumably from the same degeneration, which six weeks earlier had caused the nose bleed. On the other hand, the insidious nature of this disease, and of its onset, is a matter of such common report, that I will not weary you with any cases illustrative of this fact.

The existence of oxaluria, or of uric acid lithiasis, is a prolific source of mischief to the kidney. The deposit of oxalates, or uric acid in the kidney, and its passage outwards, occurs most frequently in persons who are large eaters, living an inert life, and taking but little exercise. Such people are common among the rich in large cities, and I have seldom failed to find tube-casts in the urine of any one who passed gravel for any length of time, whether oxalates, or uric acid, even if the calculi were microscopic in size, and this, commonly before any albumen was to be found. Later, if there is any considerable amount of gravel passed, albumen and blood will be found in the urine. If the attacks of gravel occur often, they are apt, at last, to be followed by Bright's disease, directly caused, probably, by the mechanical irritation of the lining membrane of the tubules of the kidney, by the sharp edges of the minute calculi passing through them.

It cannot be proved by statistics, and yet I think there is strong ground for believing, that the proportion of deaths, caused by affections of the kidney, is greatly on the increase. The tendency of modern life seems to be such, that a larger number of deaths are caused by degenerative processes, and less by acute disorders. In former times, children were less delicately reared, and a larger proportion of the frailer

ones probably died from exposure and want of care; now, beginning at childhood, and continuing up to old age, we are much more careful to avoid exposure, and are more self-indulgent, at least as far as our bodies are concerned. Who, in modern times, except the professional athletes, would undertake the physical labors of the Emperor Charles V. of Germany, of whom Motley says, that he was able to tell, at a time when all journeys were made on horseback, or by boat, "of his nine expeditions into Germany, six to Spain, seven to Italy, four to France, ten to the Netherlands, two to England, as many to Africa, and of his eleven voyages by sea!" Nowadays, the majority of city men are in their places of business all day, and take but little exercise, eating as much, and as rich food, as in former times, and drinking not a little withal. The natural and necessary consequence follows, that after years of this kind of life, the excretory organs degenerate: first the kidney, which is the common sewer of the body, and, like the heart, has to do continual work, from the hour of birth until the hour of death. It is a common thing, in modern life, to see a man naturally robust and gifted with a large appetite, become more inert as years go by, when he will gradually give up his active exercise, until finally he takes none: still the eating and drinking go on, and he becomes fat and sluggish; finally he loses health, and then, examination shows that degeneration is beginning in the kidney. I could instance several cases of the kind but my space will not allow me.

The drift of my paper has been to try to establish, that we should, in all ordinary cases of albuminuria, be very guarded in our prognosis, except when it is

self-evident that the case must be rapidly fatal. I have now under observation fifteen persons, seventeen, if I count two who have albuminuria alternating, or parallel with, diabetes mellitus, who have been suffering with this complaint for a greater or less length of time. With three of them, the disease began nearly nine years ago, and they either have it now, or have entirely recovered from it, after more than two years' continual duration; yet in regard to no one of them do I feel able, even in my own mind to guess, much less to formulate an opinion, as to how much longer they may live. There are absolutely no data, if such cases are considered in the light of the experience of the sixty-two I shall mention, upon which to found a positive prognosis.

Another observation I have made, is in regard to the deaths of very old people. We are all agreed I presume, that when a very old person dies, and we report the death as one of old age, it is because we are unable to put the finger upon the exact part of the old and worn out machinery, that at last refuses to work, and thereby causes all the rest of the mechanism to stop. This direct cause, if sought for, will frequently be found in the kidney. To examine the urine is not always a thing that strikes us, particularly when absolutely no symptoms appear to point toward kidney disease. I have seen a number of instances, in which old people seemed to be fading out, as they often do, yet have felt entirely unable to explain to myself any direct cause for the failure, until the urine was examined, when there was found albumen, with granular and hyaline casts, and sometimes a few blood corpuscles. A careful inquiry

would perhaps reveal the fact, that the amount of urine passed was very small, all this without any other symptom to point toward kidney disease; at the same time, the failure of the patient being very gentle and gradual; or else, as not infrequently has happened, senile delirium existed, either violent or mild in type, followed at last by death.

In the whole sixty-two cases, which I will now summarize collectively, there have occurred twenty-six deaths, thirty-four are, to my knowledge, still living, some in better and some in worse health, and of two, I have lost sight.

Five were cases of ordinary acute desquamative nephritis, with the occurrence of one death, and four recoveries. In one instance, a child, there were two attacks, two years apart, the first being unaccounted for, the second following scarlet fever; the second case occurred after scarlet fever; the third after diphtheria; the fourth was in a young man apparently healthy previously, and now in good health, his attack only lasted a short time; the fifth and fatal case was in a boy of about fifteen years of age, and came on while he was at boarding school, the onset being insidious, and the disease progressing from bad to worse, until death occurred.

Four cases seemed to be caused by the passing of gravel, principally oxalates. These people are still all living, although they have had albumen and casts present in the urine, on and off, for varying lengths of time; two of them for six years past, one for seven years, and the fourth has had two attacks of nephritic colic, about a year apart; on each occasion, albumen

and granular casts, uric acid, and blood corpuscles were found in the urine.

Twenty-two cases I class as of ordinary Bright's disease, there being probably contracted kidney in the majority of instances, and of them, fourteen are dead. I made no post-mortem examination in any one of these cases. In most of these cases (seventeen of them) the onset was insidious, there being no illness to mark the beginning of the disease; in one, the patient was seized with a convulsion at the supper-table, having been supposed previously to be in good health; in a second, there was a violent attack of congestion of the lungs, with terrible dyspnœa during the night, the patient, a woman, having gone to bed thinking herself in good health; in a third, there was dyspnœa and congestion of the lungs occurring suddenly; in a fourth the condition of the kidneys was discovered concurrently with an attack of catarrhal pneumonia; a fifth was a man who had had attacks of nephritic colic. He had a violent attack of congestion of the lungs, with bronchitis and dyspnœa, and was found to have albumen and casts in the urine, which condition continued, on and off, for four years; he is now in apparently good health, although the first attack was nearly nine years ago. Of these twenty-two cases, two were in one family, one soon dying, and of the other, I have lost sight; two in another family, one dying and the other, I think, being still alive, although the disease was discovered three years ago; five cases occurred in another family, the father, son, and three daughters. The father and mother of this family had ten living children, five reaching adult life, one died of consumption, and the other three, and the father, of Bright's

disease, the surviving daughter has had the disease for nearly nine years. In another family there were three cases, all women: one died, having had uræmic convulsions, and the other two still live, both having had the disease for about three years. A brother has now cancer of the rectum. The mother is still alive, and although seventy-four, enjoys good health. The father died at quite an advanced age, but I do not know of what disease.

The next twenty-two cases I must classify as unaccountable; eighteen of them occurred in persons over sixty years of age, and the other four were well on in life; one was over ninety, three over eighty, and two over seventy years of age. Of these people only five are dead, and of the five fatal cases, one was the man over ninety, two were over eighty, a fourth was seventy-seven, and the fifth sixty-seven.

The next four cases were of people advanced in life, and they died of general break-down, having in three instances concurrent disease of the heart and kidneys, with implication of the lungs, and the fourth had renal and brain disease.

In two instances there has been albuminuria with diabetes mellitus: in one, sugar was discovered in the urine six, and in the other four, years ago. Since then there has been sometimes sugar, and at others albumen; at times both at once in the urine of these two women. In neither instance have tube-casts ever been found. I mention these two cases in a paper upon albuminuria, because both have frequently had albumen in the urine, and I confess myself unable to explain the phenomenon, the quite frequent presence of albumen in the urine, without tube-casts, or any

other sign of renal disease, and yet I can hardly believe the albumen to be merely due to cystitis, for if that were so, it should be constantly present.

In one instance, I have followed for four years the history of a man who drinks to excess. At the beginning of, and for a time before, his attacks of delirium tremens, while he is consuming alcohol freely, he often has bloody urine, which will contain tube-casts of all the varieties at different times, fatty, hyaline, and granular. When he ceases his consumption of alcohol, as he has quite frequently done for two or three months at a time, the urine becomes normal, as far as chemical tests and microscopic examinations will reveal its condition. In the light of this case, how can we believe that the abuse of alcohol does not produce renal degenerations? Is not this man sure to die of Bright's disease, if he continues his indulgence in alcohol?

Another case was a man over fifty years of age, who was seized with rheumatism; he progressed badly, and was much more sick than the amount of rheumatism would seem to warrant, and at last, after about three weeks, died; having obstruction of the bowel and inflammation of the kidneys, as shown by the presence of albumen and tube-casts in the urine; also hydrothorax, and extreme stupor, which seemed uræmic. This man had had an attack of influenza about three or four weeks previous to his fatal illness. Can there be any connection between the severe influenza, and the rheumatism, and renal inflammation and obstruction of the bowel, of which this patient died?

The last case I will mention was a boy, who died very suddenly of congestion of the lungs, probably

the first stage of pneumonia. A trace of albumen and granular casts were found in his urine, which was obtained a short time before death. This boy was about sixteen years of age, and did not seem much sick on the afternoon of one day, being up and dressed, the next morning dying, in a state of maniacal delirium, with his lungs extremely congested. There was, unfortunately, no post-mortem examination made. This case would seem one in which the pulmonary affection was primary, and yet it might be that he had a latent contracting kidney.

There has been much said of later years about kidney disease without the presence of tube-casts in the urine, and of the presence of casts without albumen. I may say that I have frequently found tube-casts in the urine when the most careful chemical examination failed to detect any albumen, and *vice versâ*, have failed to find any casts at all, when the event proved the existence of renal disease.

The subject of treatment I have not even mentioned in my paper, although much might be said about it, because I have already trespassed too long upon your time.

In conclusion, I will briefly recapitulate the points which it has been my endeavor in this paper to prove.

First, that in no ordinary, uncomplicated case of Bright's disease, should a prognosis of speedy death, or even of incurable disease, be given, for I have related cases in which the disease was chronic, lasting more than two years, and which ended in complete recovery, and others in which the person affected has lived nearly nine years.

Second, that dyspnœa, usually taking the form of

renal asthma, is much more common than is usually supposed, and, when properly appreciated, is a valuable diagnostic sign of the disease; also that severe coryza is a complication or accompaniment, and has a diagnostic value.

Third, that Bright's disease, as a cause of death, is on the increase.

Fourth, that it is a very common cause of the deaths of old people, probably being the direct cause in many deaths reported as of old age.

Fifth, that the passage of gravel, even when microscopic in size, but particularly if large enough to cause nephritic colic, is a prolific cause of the disease.

Sixth, that the occurrence of tube-casts in the urine, without, or in advance of, the presence of albumen, is very common, and *vice versâ*, persons may die of Bright's disease, and the most careful examination fail to show any tube-casts, although there may be albumen constantly present in the urine.

Seventh, that the abuse of alcohol is certainly a cause of kidney disease, as proved by the case I have related, in which it has, again and again, caused hemorrhage from the kidney, with the temporary presence of albumen and tube-casts in the urine, disappearing again with the cessation of its consumption.

[After the reading of the preceding paper, the following remarks were made:—]

Dr. R. P. Harris said that he had observed, in cases of albuminuria, vomiting as a frequent symptom, and regarded it of diagnostic importance.

Dr. H. C. Wood said he desired to ask with regard to the diagnosis of Bright's disease in cases in which neither albumen nor tube-casts are found on examination of the urine. In a case of this character, which had come under his observation, he based the diagnosis on the low specific gravity, and the diminution in the quantity of urine secreted.

Dr. J. Solis Cohen said he had long been aware of some special associations between laryngeal and renal diseases, and had attributed this to disturbance of circulation at the base of the brain, affecting both the renal nerves and the pneumogastric.

Dr. Bruen called attention to the prognosis of catarrhal diseases of the kidney, classed with Bright's disease. When these forms of renal disease were associated with severe catarrhal lesions in the lungs, the renal disease accelerated the fatal issue.

But sometimes catarrhal lesions may be regarded as a local expression of general catarrhal inflammation. The etiology of renal disease offers most favorable vantage to the physician for basing his prognosis. The prognosis is good in these cases when the heart is hypertrophied and an adequate circulation is maintained; bad in proportion to advance of cardiac mischief. Prognosis, then, should be made only for six months at a time. In *Guy's Hospital Reports* for 1881 are to be found a number of cases of Bright's disease without albuminuria by Mahomed. Dr. Bruen called Dr. Wood's attention to these cases, and expressed a belief that a close study of the sphygmograph tracings would be of great assistance. Further, the character of the cardiac sounds indicate the strength of the heart. This arterial fibrosis, with cardiac hypertrophy, accompanies most markedly the forms of Bright's disease in which the interstitial changes predominate over the tubular. The urine in these cases, though it may not

exhibit albumen, will often exhibit casts, mostly hyaline slightly granular.

Dr. Bruen called attention to the value of the shallow cell in the examination of the urine, when the casts are but few. This method is highly recommended by Dr. Johnson, and Dr. Bruen desired to elicit the experience of others as to the necessity of examining urine in a small cell in all doubtful cases, when casts might be supposed to be at a minimum.

Dr. James Tyson said the subject of albuminuria was so extensive and presented so many phases that one is at a loss where to commence in its discussion; but he believed Dr. Meigs quite right in saying that the more one studies it the more unwilling one is to interpret it dogmatically. He thought, however, that there are some cases in which a quite positive position can be taken. To illustrate: with contracted kidney or interstitial nephritis, he thought the presence of hypertrophy of the heart a symptom which justifies an unfavorable prognosis, while in cases of contracted kidney in which this stage has not been reached it was most difficult to approximate the time of termination. He had seen persons apparently at the point of death recover so far as to be able to resume their occupations, while others, apparently in the vigor of health, succumbed in a few days with uræmic symptoms. Generally death soon succeeds upon the occurrence of uræmic convulsions, and Dr. T. could not recall a case in which they were not followed by an early death in this form of kidney disease.

Again, in cases of chronic parenchymatous nephritis where there are large numbers of highly fatty cells, oil casts, and free oil drops, while the patient exhibits a pallid whiteness which suggests the almost total absence of red-blood corpuscles, recovery is scarcely possible. On the other hand, he had seen many cases in which oil casts were in the urine recover completely.

Dr. Tyson could not, however, accept the quotation by Dr. Meigs, from Grainger Stewart, as expressing the exact truth. It is not strictly true that every form of cast may be found in every form of Bright's disease. Blood casts are not found in uncomplicated chronic Bright's disease; and, although it is common to find hyaline casts containing a few oil drops in chronically contracted kidney, highly fatty casts are never present. He did not desire to be considered as saying that casts are of conclusive value

in diagnosis, but in many cases they are of great assistance. It is more particularly in the subacute and chronic forms of parenchymatous nephritis and amyloid disease that we have the same varieties of casts present, and find them consequently of little value in diagnosis.

Much is said nowadays of albuminuria in health. But Dr. T. thought that however complete may be the absence of symptoms accompanying an albuminuria, he could not consider the kidney from which filtered a continuous albuminous urine to be in a normal state.

With regard to the question of Dr. Wood he thought that even throwing out those cases in which albumen and casts are overlooked by the observer in consequence of carelessness, haste, or inexperience, it was possible that there might be Bright's disease without either albumen or casts. But he had never met an instance. In such a case he would consider the symptom mentioned by Dr. Wood—low specific gravity along with a greatly diminished urine—one of the most conclusive possible.

Dr. George C. Harlan spoke of the great importance of an examination of the eye-ground in Bright's disease, and of its value in a diagnostic point of view.

Joseph G.

Dr. Richardson suggested that there was one point in connection with the prognosis of Bright's disease, briefly alluded to in Dr. Meigs's excellent paper, which might prove worthy of more extended study. Some years since he had published an article, calling attention to the value of the red corpuscles of the blood in the urine of Bright's disease, as indicators of the form and stage of the affection (vide *American Journ. Med. Sci.*, Jan. 1870, p. 54), and he believed that where these corpuscles were found, as they might often be by a sufficiently careful examination, attached to, or imbedded in the casts, they were elements of great prognostic as well as therapeutic importance.

Dr. Arthur V. Meigs, in closing the discussion, said that his paper was purely clinical, and was intended merely to record observations made in studying the sixty-two cases of albuminuria mentioned. It would self-evidently be impossible, in so short a compass, to attempt to give even a *résumé* which would be exhaustive of so vast a subject as the one under discussion. With

regard to the observation of Dr. Wood, that Bright's disease might exist without either albumen or tube-casts being present in the urine, he thought it a fact which could not be denied. As to the possibility of making a diagnosis under the circumstances, the observation of Dr. Wood that a small amount of urine of low specific gravity passed continuously for a length of time, and that of Dr. Harlan that the ophthalmoscope might sometimes lead us to a positive conclusion, seemed at present our best, if not only means of making such a diagnosis. Vomiting is a common accompaniment or symptom of renal disease. He agreed entirely with Dr. Tyson, that in a certain class of cases of Bright's disease a very exact diagnosis was possible, and a prognosis too; but in other instances, particularly in patients advanced in life, and with the disease somewhat latent, even an approximation of the future duration of life was impossible.

AUTOPSY OF A CASE OF TRANSPOSITION OF THE VISCERA.

By

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[Read October 4, 1882.]

The comparatively rare occurrence of the phenomena, an instance of which I am about to relate, will be, I think, sufficient excuse for occupying the time of the College. I regret that I am not able to offer a more complete record of the case; but a careful dissection was impossible without taking an unwarranted risk of septic poisoning, as the abdominal cavity was filled with fetid pus.

Frank M., aged 21, died at the Presbyterian Hospital of purulent peritonitis. I shall ignore in this paper the pathological conditions found at the post-mortem examination, inasmuch as they have no bearing upon the subject under consideration.

The patient had been aware since boyhood that his heart was misplaced, and upon one occasion a physician told him that that was the reason he was left-handed. He had never been very strong; but it does not appear that he ever had any serious affection; the cause of which could be assigned to his misplaced viscera.

In 1878, when he first came to the hospital, Dr. James Markoe, under whose care he was, noticed that not only was

his heart perceptible in the fifth interspace of the right side, but also by percussion ascertained that this organ occupied the same relative position upon the right side that it should upon the left. He found also that the liver and spleen were transposed, but no further changes seem to have been noticed.

At the time I made the post-mortem examination, I found externally nothing to indicate the great changes in the positions of the internal organs, except that the right testicle was suspended at a lower level than the left. When I had opened the thorax and abdomen and laid bare the organs contained therein, the appearance to me was as though I was looking at the body reflected in a mirror. It was like the photographer's negative.

The heart first attracted my attention, and was found to be normal in every respect, but occupying, as already noted, a position upon the right side. It looked as though it had been pushed over to its present position without changing materially its relations with other organs. The right side was the pulmonary side and the left the systemic side, and in this respect the organ performed its function in a normal manner. The aorta starting from the left ventricle followed a somewhat irregular course, crossing over the right bronchus and going from there to the right side of the vertebral column, along which it descended upon the right side. The branches given off from the arch of the aorta were the two coronary arteries unchanged, the innominate going up the left side of the neck and giving off the left common carotid and left subclavian. While coming from the right portion of the arch were the right common carotid and right subclavian.

The lungs were reversed. The right lung was found to be smaller to allow space for the heart. It had two lobes.

The left lung corresponded in appearance to what is commonly the right lung, was shorter than its fellow, being pressed up by the liver, was broader, and had three lobes.

In the abdominal cavity the liver occupied the left hypo-

chondriac and partly the epigastric regions, the large lobe to the left and the smaller ones to the right, the gall-bladder and everything else similarly changed.

The stomach and intestines were transposed. The stomach was partly in the right hypochondrum and partly in the epigastric region. The pylorus extended forward and downward toward the middle of the abdomen. The spleen was on the right side, and attached to the greater curvature of the stomach.

The cæcum was situated in the left iliac fossa, and the colon ascended on the left side and crossed from left to right side of the abdomen. From there it descended and terminated in the sigmoid flexure upon the right side.

As already stated, the man was naturally left-handed. Hyrtl was the first, I believe, to advance the theory that we are normally right-handed because of the more direct flow of blood to the brain upon the left side. And this theory would be substantiated if all cases where the heart and great vessels were transposed were left-handed; but such is not the case. A few years ago a theory was advanced that right-handedness depended upon natural selection. But the easiest explanation is that from earliest infancy we are instructed to use the right hand always in preference to the left. When a person with transposition of the viscera is left-handed we may call it a coincidence. In any other view, it would be extremely difficult to account for the far larger number of persons whose organs are arranged normally and yet are left-handed. In the cases that are noted in the journals I have not been able to find statistics enough to draw any inferences, for, when this condition is noted, right-handedness appears to be as fre-

quently met with as left-handedness in cases where the viscera are transposed.

Peacock, in his monograph on Malformations of the Heart (T. Peacock, *Malformations of the Heart*, 2d edit. 1866), says that the most common of all the internal displacements is that in which the heart is placed upon the right side in a position corresponding to that which it should occupy upon the left, and that this displacement is almost always associated with more or less complete transposition of other viscera. Occasionally the heart is transposed, while the other organs are unchanged (Sur l'Ectopie de l'appareil de la circulation et particulièrement sur celle du cœur.—*Rep. Gen. d'Anat. et de Phys. Pathol.*, t. ii. 1826, p. 1).

Among the earliest recorded cases are those of Servius and Schenkius in Rome in 1643 (Thomas Bartholinus, *Hist. Anat. Cent.*, ii. 29. Amstelodami, 1654), and one by Riolan in 1652 (Allen Thompson, *Glasgow Med. Journ.*, vol. i. 1853, p. 220).

The first case recorded in which the malformation was noticed during life appears to be that described by Morand (Méry, *Memoirs of Academy of Paris for* 1866) in 1666. This case seems to have attracted considerable attention at the time, and was said to have suggested to Molière the transposition of the heart and liver which he puts into the mouth of Sganarelle in his play of *Mèdecin Malgré lui*, published about the time this case was reported.

In cases of more or less complete transposition, the arteries, if they do not retain their natural positions, may be displaced in almost all conceivable ways.

Dr. Gamage (*N. Eng. Journ. Med. and Surg.*, iv.

1815, p. 241) reported a case in which the aorta arose from the right ventricle and the pulmonary arteries from the left ventricle. Mr. Abernethy (*Philosophic Trans.*, 1793, p. 59) saw a child, ten months old, where the portal veins terminated in the vena cava, the liver being supplied from an unusually large hepatic artery. Mr. Douglas Fox (*Lond. Med. and Phys. Journ.*, vol. li. 1824, p. 474) reported the result of a post-mortem examination in a case where the heart was transposed, and the aorta, after crossing the right bronchus, passed behind the lower end of the trachea, over the vertebral column, and pursued its usual course from there down the left of the spine.

For a very complete account of a case similar to the one I now report, and for an exhaustive study of the histology of such malformations, I will refer to Dr. Allen Thompson's paper in the *Glasgow Medical Journal*, vol. iv. p. 220.

As regards the practical application of this case, it would be well to bear such abnormality in mind as a possible condition in cases brought to a physician's notice in an unconscious condition. It will readily be seen that considerable difficulty might arise in forming a diagnosis. In surgical operations upon thorax or abdomen it would prove a serious complication, as, for instance, in colotomy, paracentesis abdominis, or pericardii.

FLEXIBLE GELATIN AS A SUBSTITUTE FOR ADHESIVE PLASTER.

By

ADDINELL HEWSON, A.M., M.D.

[Read October 4, 1882.]

In the *Boston Medical and Surgical Journal* for October 7, 1880 (vol. ciii. No. 15), there was published under the caption of "Original Articles" a paper which I had previously read at a meeting of the Academy of Surgery in Philadelphia on "Substitutes for Adhesive Plaster." In that paper I had taken the broad ground of objecting to all forms then known of such plasters from the fact that they all—even Prof. Lister's—contained more or less of resin which had long been recognized by a majority of experienced surgeons as a source of irritation to the skin, and an actual cause of erysipelas in many instances. This latter circumstance, even only as a possible result, was a sufficient cause for our discarding all such plasters.

In that communication I referred to various devises, especially to the mode of using Maynard's Collodion as proposed by the late Dr. Paul Beck Goddard, of Philadelphia, one of the greatest geniuses of his day and generation in our profession. It consisted of using strips of Donna Maria gauze as a medium for

collodion and skin to adhere together and so bind the wounded parts as plasters are meant to do.

This gauze Goddard cut into strips lengthwise of the tissue, of the desired width and of a length two or more inches in excess of what would be needed to fasten the gauze for an inch on each side of the wound; the latter expedient was to facilitate whatever traction might be needed to effect complete coaptation and adjustment of the edges of the wounds after the remote end of the gauze had been secured, as was to be desired, and the time had arrived for securing the gauze on the near side. When the latter was accomplished, the parts immediately under the gauze and which were involved in the wound, could be plainly seen and determined to be correctly fixed, and then secured by the painting on the collodion as on the other side.

Nothing, then seemed to me, could be more desirable than this use of collodion as made in the neat and dexterous manner of Dr. Goddard, and I can confess boldly and candidly that I have never been, since his first demonstration to me, without the details of his dressings or those of one essentially like his. For it is true that I have used the common tarletan of the trimming stores in place of the more expensive silk tissue, and that I have more recently had myself constantly provided with liquid glue, made with acetic acid and common glue, in place of the collodion, as the collodion since then has been generally made with a small portion of resin or of turpentine to prevent the film, formed by its drying, from cracking or pealing off, a component which made it as objectionable as the emp. adhesiv. This liquid glue, which

I was thus led to use instead of collodion, is very readily made. It consists of one part by weight of glue made liquid by melting in a hot water bath, the ordinary glue pot, and when so melted four parts of officinal (2.5 per cent.) acetic acid are to be slowly stirred into it. This quantity and quality or strength of acetic acid are essential to make a perfectly smooth fluid glue, and one that will dry as quickly as collodion. There is a smell about such a compound caused essentially by the acid, which can be readily covered by a few drops of the attar of roses—these should be as few as possible, for they are actually drops of an essential oil, all of which we have to dread, as we have turpentine, on account of being liable to induce erythema, erysipelas, and the like. This addition of perfume to the liquid glue may sometimes be needed where cerates and the like are used as topical applications, but I have never had to resort to it, as long before I had abandoned, or sought for a substitute for the collodion, I had become constant in the use of dry earth for such topical applications, and had learned that it was alike a deodorizer as well as a disinfectant on such occasions. The objection was, nevertheless, a good one against the glue, and although I have been using it for over three years I have never been satisfied that I had attained to the production of an article, or of a combination of articles, which, whilst they fulfilled all the *desiderata per se* of a perfect sticking plaster, were not open to some objections.

Starting with the conviction that our adhesive material had to be fluid to use it with the gauze tissue, I labored long in that direction for something free from the objections to the glue. An incident after a time

led me to question that conviction; this was a demonstration by a mountebank, of the possession of a piece of solid mucilage with which he could glue together by their edges the fragments of a bank note, which he had torn in pieces before my eyes. He took his piece of solid mucilage and moistened its free end with saliva from his mouth, and when he seemed to have got it sufficiently dampened, he arranged the torn edges, and so rubbed the mucilage on them, and after wiping clean the surface of those pieces, he laid them on a clean piece of paper so that their torn edges would come in the closest juxtaposition, and then rubbed them there by a piece of clean blotter. On the removal of the latter one could readily see that the torn edges of the note were neatly adherent to each throughout. Then, after lifting the note off the table and making strong traction on its extremities and in the direction of its surface, it was evidently imposible to make them give way at any one of its glued parts. Here was the kind of article I was seeking for. Speaking of the subject to a patient, who held a prominent position in a bank, he informed me that they used some such substance for mending notes in their establishment, but that it smelt very badly. Meditating over these facts I determined to take the subject up at once, and consulted Mr. Ottinger, a druggist of this city whom I knew to be a man of zeal, very ready and free with the use of his knowledge. He took hold of the subject at once and agreed with me as to the propriety of trying what could first be done with the common glue. I was familiar with the effects, as he was, of glycerine in making glue flexible without diminishing its tenacity or adhesiveness, and

we decided to experiment first with the white glue and English glycerine. This was in the hot weather at the beginning of September of this year. The experiment was made with half a drachm of glycerine to one ounce of the melted glue. This made the glue very slow in drying enough to make a stiff mass, and other experiments showed that the amount of glycerine necessary was really very trifling. We have finally reached the conclusion, that the addition of twenty-five drops of glycerine to the ounce of melted glue, would make a mass sufficiently flexible in cold weather, and adhesive enough for our purpose.

This mass Mr. Ottinger has made for convenience into quadrangular cakes one inch long, five-eighths broad, and four-eighths thick, and to preserve their flexibility he has enveloped them in one or two thicknesses of waxed tissue paper, such as I have long been using in place of oiled silk. One end of these cakes is always left uncovered and can be made ready for use by dipping it in hot water, and then rubbing it freely over the surface on which the end of the gauze is to be applied. The form and size which has been adopted, I suggested as that which would be required for each individual case of amputation.

One box contains not only one but four of such cakes, and also enough of the tarlatan as will be needed in a case of major amputation until it is healed. By adopting the quadrangular form and the differences in the length, breadth, and thickness we secure greater facilities in using the stick than would occur with any other form; they first of all give us decided advantages, for firmly holding the stick, over what we can get if it was rounded, then we can make the glue

penetrate into corners or close cracks of the part and rub it through the meshes of the gauze wherever we may subsequently desire it fixed over again. A rule should be adopted, in cases of amputations, to leave the cake at the patient's house, cleansed after each time of its being used, and put away well protected until again needed.

The cake should be thrown away when no longer needed in the case where it was first used. If it has been very much used and so become too short for ready handling in the case, I prefer taking a new piece rather than running any risk of contamination by using dressing forceps—a large needle or the like, as a ready holder for that remnant of the piece originally used.

In the dressing of trivial wounds I have myself always prepared against waste by having had one of the cakes divided lengthwise into four pieces—each one of these is ample to serve such a case generally from beginning to end. They should be enveloped in wax paper as the cake was originally, and are to be kept and then discarded as directed for the original cakes. The division of the cakes into four is all that may be made of them—for dividing them again lengthwise, which is the only further division, would make them too short for handling with facility.

This plan of securing each patient with his own piece from the very first dressing is a most valuable one. It looks at first like great extravagance to throw away a piece before, in many instances, it has been used a half a dozen times; but it really costs so little that further saving need not be considered. A box of this dressing can be got for twenty-five cents, and

it contains four original sized cakes, with enough gauze (thirty square inches) to serve the dressing of a stump throughout. The actual cost of this gauze is ten cents a square yard; making what is furnished in the box, enough for the stump, cost less than the one-fifth of a cent. Charging then the cost of the whole box to the four cakes of glue, the cost of the adhesive dressings of a stump will be less than seven cents, and for those of a seriously lacerated or incised wound, less than two cents.

REPORT UPON A SPECIMEN OF XANTHIC OXIDE CALCULUS.

By

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[Read December 6, 1882.]

I DESIRE to present for Dr. George L. Porter, of Bridgeport, Conn., a specimen of xanthic oxide calculus. It consists of one-half of a stone, an inch and a half long and an inch wide. This half is to be deposited in the Museum of the Jefferson Medical College, the other half is in the Army Medical Museum, Washington, D. C.

So rare is this calculus that, including the present specimen, only eight have ever been described, and none of them so completely as Dr. Porter's. Moreover, this is the only specimen ever recognized and described by an American surgeon. Four of the specimens are British, two French, and one German. Marcet in 1817 described the first calculus of this kind which was recognized.

The present stone was passed spontaneously by a woman eighteen years old. Its clinical history presents nothing specially worthy of note, but its chemical constitution makes it very interesting. Xanthine or xanthic oxide is analogous to uric acid, having, however, one less equivalent of oxygen, and

is the rarest of all calculi. In the *New England Medical Monthly* for May, 1882, Dr. Porter relates the case in full, with a drawing of the stone, and an analysis and comparison of the eight cases on record and some interesting remarks on xanthic oxide itself.

[After the reading of the preceding paper:—]

Dr. JOHN B. ROBERTS stated that in 1873, Dr. R. J. Levis operated for vesical calculus by lithotrity on a man. The patient, who was aged 69 years, was an inmate of the Pennsylvania Hospital. The fragments were examined by the late Dr. H. B. Hare, the well-known pathological chemist, and found to consist of xanthic oxide. The patient was discharged by request of his friends, while some of the stone still remained in the bladder, and passed from the surgeon's observation.

Dr. JAMES TYSON said that in connection with the case just reported by Dr. Keen, he desired to place on record a case which came under his own observation, of persistent cystin sediment in urine, concurrent with impacted oxalate of lime calculus. G. B. W., a very intelligent physician residing in one of the southern counties of Pennsylvania, and 45 years of age when he first saw him, was lithotomized in Baltimore when he was 28 years old, and a calculus of pure *cystin* removed. From that time he continued, according to his own account, to pass cystin daily. Early in January, 1879, a specimen of urine was sent to Dr. Tyson in which there was considerable pus and a proportionate amount of albumen. In this specimen there was found no cystin, but in later specimens there were found large numbers of the characteristic crystals along with pus and albumen. A little later Dr. Tyson visited him at his home, and found him suffering greatly with extreme lumbar pain, attacks like which he had frequently had before, but the present was one of unusual duration, and had greatly prostrated him. There seemed every reason to believe there was a calculus impacted somewhere between the left kidney and the bladder. His sufferings continued, and he was only relieved by death, which occurred on the 6th of March, 1879.

The following notes of the autopsy were received from Dr. Wm. B. Rowland of Rowlandsville, Md.: The *post-mortem* examination revealed a calculus in the left ureter just ready to pass into the bladder. The calculus was the size of a large pea, and very rough. Just behind where the calculus was found in the ureter was a collection of pus, dipping down into the pelvis, which would soon have made its exit through the ischiatic foramen if life had been prolonged.

The left kidney was somewhat enlarged, and the right was not more than one-fifth the usual size, but apparently healthy. The bladder, stomach, and bowels were healthy. No mention was made by Dr. Rowland of the liver, which was presumably healthy, but there was found in the gall-bladder a calculus an inch long and half an inch in diameter throughout its length.

The calculus which is exhibited to the College presented none of the physical and chemical characters of cystin calculi, which are smooth and friable, but is evidently oxalate of lime. This is particularly interesting in view of the fact that a cystin calculus was removed by lithotomy seventeen years earlier, and that the patient so persistently passed cystin crystals up to the time of his death.

A RÉSUMÉ OF TWENTY-FIVE CASES OF ABDOMINAL SECTION.

By

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[Read December 6, 1882.]

With a view of placing on record the results in a number of cases of abdominal section, and with the hope of contributing to the information possessed already with regard to these operations, I beg to submit the following *résumé* of the cases which have come under my care.

I have endeavored to present the points, which were regarded of interest, in a concise manner, for this purpose grouping them under different headings.

Of the twenty-five cases of abdominal section, twenty-two were performed for the removal of tumors of the ovary; one was made in a case of encysted dropsy of the peritoneum (reported in the *Transactions* of this College, 3d Series, Vol. I.); one in a case of abdominal dropsy, in which the diagnosis was obscure, and an operation of exploration was made; and one for removal of the child in extra-uterine fœtation.

As the case of encysted dropsy has been reported

to the College, and as I propose, at a future time, to report the one of extra-uterine pregnancy, I shall present on this occasion a *résumé* of the ovarian cases.

Age of Patients.—In the twenty-two cases the age varied from the youngest, sixteen, to the oldest, sixty-five years.

Nationality.—Sixteen patients were natives of the United States, and six of Ireland.

Social Condition.—Five were single, one was a widow, and sixteen were married.

Duration of Growth.—The duration of growth varied from three months to seven years, counting from the time at which the tumor was recognized first by the patient.

Aspiration, or Previous Tapping.—Aspiration, for the purpose of obtaining a specimen of fluid for examination, was performed in eight cases; tapping, to relieve abdominal distension, in four. In one case, in which the cyst was very fully distended by fluid, and the abdominal wall was very tense, leakage followed aspiration, and persisted for some hours, despite the efforts made to control it by pressure. In none of the cases, in which aspiration or tapping was performed, did any serious results occur, nor were there any evidences in the operations, which followed, of complications due to the previous aspiration, or tapping. In all cases, proper precautions were taken, the patient being required to rest in bed from three to four days subsequent to the operation.

Condition of the Patients at the Time of Operation.—With the exception of two, who were the subjects of malignant disease of the ovary, the condition of the

patients was good. All were placed upon preparatory treatment, extending over periods varying from two weeks to two months.

Line of Incision.—In all cases the abdominal cavity was opened by incision in the linea alba, midway between the umbilicus and pubes, the length of the incision varying according to the nature of the tumor and the presence or absence of adhesions. A simple, mono-cystic, non-adherent growth was extracted easily through an opening one and a half inches in length; whilst other tumors, polycystic in character, with numerous and strong adhesions, required incisions from four to six inches in length in order that the hand could be introduced into the abdominal cavity, so as to sweep over the external surface of the tumor for the purpose of detaching adhesions, and also into the interior of the growth to disintegrate its contents, and thus reduce its size. The incisions were invariably closed by the introduction of metallic sutures—iron or silver wire—the needle being carried so as to include the peritoneum.

Adhesions.—In eighteen cases adhesions, either parietal, omental, or visceral, existed—differing greatly as to extent and character. In some instances they were so slight as to be separated readily with the finger; in others, they were very extensive and very firm, requiring some force to effect their detachment, and exposing denuded, bleeding surfaces. In one of the fatal cases the adhesions were universal, and so firm as to require a minute dissection to be made, in order to effect removal of the cyst.

In a second case, a portion of the parietal surface of the peritoneum as large as the palm of the

hand, and embracing the sub-peritoneal fascia, was detached, leaving a broad band of attachment. This was included in three animal ligatures, and the detached portion removed. During the period of recovery, which was not retarded, the patient referred to the position of the exposed surface as a sensitive point. In still another case, an adhesion, in the form of a cord, at least two inches in thickness, and from four to five inches in length, fastened the tumor to the parietes. It was drawn down and a double animal ligature applied before section was made. Hemorrhage caused by the separation of adhesions has been controlled by the application of carbolized silk and animal ligatures, both ends being cut short, and the ligatures permitted to remain *in situ*. In some instances, torsion of the vessels has been sufficient to restrain the bleeding.

Character of the Cysts.—Four cysts were unilocular, sixteen were multilocular, and in two, malignant disease existed. In one of the latter medullary cancer involved both ovaries, and in the other colloid disease was present.

Double ovariotomy was performed in two cases.

Primary or Secondary Operation.—In one case the operation was secondary, ovariotomy having been performed thirteen years previously. In this instance the incision was made to the side of the cicatrix of the primary operation, so as to avoid wounding the pedicle of the tumor removed, which was found to exist as a small cord attached to the inner surface of the abdominal wall, and to be of such length as to permit the uterus to occupy a normal position. Elongation and shrinkage of the pedicle has been observed

in *post-mortem* examinations made in cases, in which death occurred some years after ovariotomy.

Treatment of the Pedicle.—In all but one case the pedicle was secured by the application of the clamp; in the case excepted, a carbolized catgut ligature was applied, both ends cut short, and the pedicle was returned to the abdominal cavity. In cases of very short pedicles I have been able always to secure them with the clamp, and in no case was it observed that the traction made to accomplish this produced any harm. In one instance of an extremely short pedicle, where, in fact, the wall of the cyst was separated not more than a half inch from the uterus, a double animal ligature was applied, which failed to control the hemorrhage. The clamp was then applied over the ligature, bringing the uterus well up between the edges of the incision. Although the patient had a tedious convalescence, the ligature and clamp came away in good time, and the abdominal incision healed kindly. Beneath the cicatrix the uterus could be distinctly outlined, and after the return of the patient to her usual duties no complaint was made of pain caused by traction upon the organ. In one case only was there noticed a slight tendency to the occurrence of ventral hernia, and this in a young patient who slipped and fell on the icy pavement a short time after recovery from the operation. A good deal of tension of the abdominal walls was felt in the act of falling, and it was thought a slight detachment of the pedicle had occurred. Rest in bed for a few days, with pressure over the cicatrix, relieved the condition.

In nearly all of the cases in which the clamp was

applied it was observed that it could be removed safely at about the same time with the last of the sutures, and, therefore, the healing of the abdominal incision was not materially delayed. In one or two cases both sutures and clamp were permitted to remain longer than usual. In favorable cases the first of the sutures was usually removed on the sixth day, and the clamp on the eighth or tenth.

In three cases menstruation has occurred by the pedicle. In all of the cases it took place but once, and did not produce any serious inconvenience. It occurred in one of the cases in which double ovariotomy was performed, two clamps having been applied to the pedicles without difficulty.

Strangulation of the intestine has been noted as being due to the use of the clamp. In my observation of the cases of others, and in my own, I have not met with an accident of this nature. As it has followed likewise the use of the ligature, it cannot be ascribed alone to the employment of the clamp.

The support afforded to the uterus by the attachment of the pedicle to the abdominal walls has seemed, in some of the cases, to have been of benefit, overcoming displacements which were productive of much previous discomfort.

In one of the three fatal cases which occurred, the ligature was applied and the pedicle returned to the abdominal cavity: death resulted on the third day from septicæmia, and the autopsy showed the stump of the pedicle softened and covered by a grayish slough. This condition of the pedicle was, I think, not a primary condition, but was a part of the general inflammation which pervaded the abdominal cavity,

occurring in a case in which the cyst was adherent in every part to the parietes and viscera, and which required minute dissection to effect its separation.

While the tendency of the present day is to return to the use of the ligature as an exclusive method of treating the pedicle, I think it unwise to discard entirely the clamp. The imbedding of the ligature and its subsequent absorption demand a degree of reparative power, which some much debilitated patients do not possess; in such cases it would appear proper to employ the clamp.

Drainage.—In one case it was thought desirable to secure drainage of the abdominal cavity after operation. For this purpose a large perforated rubber tube was introduced and allowed to remain in position for three days. During this period no fluid escaped, and the symptoms presented by the patient gave no indication of the collection of septic fluids. Of the great value of drainage after ovariotomy there can be no question. Its use is especially indicated in cases in which adhesions of some extent have existed.

Antiseptic Precautions.—In fourteen cases the antiseptic methods were employed in full detail at the time of operation, and partially during the conduct of the after-treatment, the spray being then omitted. The successful results which have attended some of the cases were undoubtedly due to its use. The condition of the patients during the after-treatment was favorably influenced, and convalescence was promoted. Of three fatal cases, one occurred after operation under the system. The tendency of most operators at the present time is to employ a modified

form of the system, owing to the fear of constitutional impressions made by agents employed. The constitutional effect of the carbolic acid has been observed in two or three cases in the condition of the urine; other than this no symptoms were noted.

It has been stated above that menstruation by the pedicle occurred in three cases. In one a marked impression was made upon the temperature, and the elevation, occurring as it did without being accompanied by a corresponding increase of the pulse-rate, attracted attention. On the day preceding the appearance of the flow the pulse was 84 and the temperature normal, $98\frac{1}{2}$°. On the day of its appearance the temperature rose to 100°, and on the third day reached $100\frac{3}{5}$°, then declined to $99\frac{2}{5}$°–99°, and on the day of the cessation of the flow returned to the normal, $98\frac{1}{2}$°—the pulse-rate in the meanwhile remained unchanged. As the elevation of the temperature occurred after convalescence had been fully declared and the patient was within two days of the period when she would have been permitted to sit up, some anxiety as to the cause existed which was not relieved until its relation to the presence of the menstrual flux was considered.

In two of the cases pregnancy occurred and terminated safely in connection with the development and growth of the cysts. In both, the cysts had attained large size, and notwithstanding the pressure exerted during parturition rupture did not occur. In one, puerperal peritonitis supervened, causing the formation of extensive adhesions; in the other, slight but firm adhesions were found.

In cases under my care recently, quinine has been

administered in large doses in the twenty-four hours preceding the operation with a view to obviate shock, and in this respect its use has been attended with success. Thirty to sixty grains, in divided doses, have been given, and in each case so treated shock has been absent. In the preliminary and after treatment it has also been given in tonic doses.

The duration of the operation has varied from thirty minutes to two hours; in the former time, mono-cystic non-adherent tumors have been removed and the wound closed. The latter period of time has been required to remove polycystic growths, with extensive and firm adhesions and many bleeding points to control. Serious complications during the operation and after-treatment have occurred in but two cases. In one already alluded to, the adhesions were so extensive as to complicate seriously the operation and to render the result fatal. In the other, the slipping of the ligature and the persistence of hemorrhage for some hours after the closure of the wound complicated the operation. This patient's recovery was slow, two months and a half elapsing before she was able to leave her bed, in which period there occurred in order the following complications—obstinate, uncontrollable diarrhœa, suppuration of hemorrhoids, formation of a large bed-sore over the region of the sacrum, with destruction of the sacro-coccygeal articulation and a condition of blood poisoning with swelling of the left parotid gland. Recovery finally took place, and the patient has been able to maintain herself by her work as a seamstress.

The size of the tumors varied greatly, and the weight from three to sixty pounds.

With one exception, all of the operations have been performed either in private houses or in a private hospital. One was operated upon in a private room of a general hospital, and in this a fatal result ensued—death, however, could not be attributed to this fact, but rather to the complications which existed in the case. In all cases careful attention was given to the preparation of the apartments so that the patients should be placed under the most favorable hygienic conditions.

In the cases in which the progress was favorable the patients, as a rule, sat up in bed on the twelfth day, and on the fourteenth were permitted to get out and occupy the lounge or an easy chair. At the end of the third week gentle exercise about the house, and in favorable weather, in the open air was allowed. This exercise was continued daily so as to prepare the patient, if living out of the city, for the journey home, which was undertaken between the fourth and fifth weeks after operation.

For twenty-four hours after the operation no food was given—at the end of this time, one ounce of milk, with lime-water, if vomiting had occurred, or if there was nausea, was given every three or four hours. In two or three days the amount of milk was increased to two ounces every three hours, alternating with a teaspoonful of beef-juice in three tablespoonfuls of water. In some cases the beef-juice was administered, instead of the milk, from the first. As convalescence advanced additions were made cautiously to the diet, no solid food being given until the sutures and clamp had been removed and the bowels moved freely by enemata.

Usually enemata of soap water and olive oil were administered on the eighth day and on alternate days, subsequently, until evacuations occurred naturally. The catheter was used every six or eight hours for five days, and then efforts at evacuation of the bladder were permitted to be made by the patient.

When possible to avoid it, opiates were not administered. When required to relieve pain or secure rest, morphia in one-sixth to one-quarter of a grain was given hypodermically.

In the twenty-five abdominal sections death occurred in four cases—three after operation for the removal of ovarian cysts, and one after operation for the removal of the child in extra-uterine fœtation. Septicæmia was the cause of death in two of the ovarian operations, and in the case of extra-uterine pregnancy. Shock and hemorrhage produced a fatal termination in the case of malignant disease of both ovaries in which double ovariotomy was performed.

The result, in the other case of double ovariotomy, was successful.

HEART-PUNCTURE AND HEART-SUTURE AS THERAPEUTIC PROCEDURES.

By

JOHN B. ROBERTS, M.D.

[Read January 3, 1883.]

It is more than probable that, in a few years, puncture of the heart-wall (cardicentesis), with direct abstraction of blood by aspiration, will be recognized as the best treatment in cases of greatly dilated or much distended right heart, with intense pulmonary engorgement; and that incision of the pericardium with suture of the heart muscle will be accepted as proper in cardiac wounds. Hence these latest novelties in cardiac surgery deserve the attention of the Fellows of the College.

That punctures of the heart are comparatively harmless has been well known to many for some years. In 1872, Roger, while performing pericardicentesis on a child with pericardial effusion, thrust the needle into the right ventricle and withdrew about $6\frac{1}{2}$ Troy ounces (200 grammes) of pure venous blood. The boy, who was aged five years, became pale, sweated, and had an imperceptible pulse. The withdrawal of the pericardial fluid, accomplished prior to the heart injury, was beneficial; and the cardiac puncture did no permanent mischief, for the patient

recovered. Death occurred five months later from long existing dilatation and valvular disease of the heart (*Bull. de l'Academie de Médecine*, 1875, p. 1276).

In Hulke's case (*Trans. Clinical Society of London*, viii. p. 169), a woman with pleuro-pneumonia was supposed to have large pericardial effusion, and a trocar was introduced through the fourth left intercostal space. Nothing escaped except a drachm of venous blood, after which the patient seemed relieved of dyspnœa. She died four weeks later from a complication of diseases, and the autopsy revealed cardiac dilatation and valvular changes.

I have said elsewhere (*Paracentesis of the Pericardium*, 8vo., Philadelphia, 1880), in commenting upon this case: "The abstraction of blood seemed to relieve the distended heart much better than phlebotomy would have done, as was evinced by the diminution of threatening symptoms and the decrease of the area of dulness."

Cloquet, Bouchut, Legros, and Onimus have also observed the apparent innocuousness of wounds of the heart made by capillary trocars. Steiner found, ten years or more ago, that electro-puncture needles could be quite safely introduced into either ventricle, provided they were at once withdrawn (*Med. Times and Gazette*, May, 1873, p. 492, from *Langenbeck's Archiv für klin. Chirurgie*).

It has been considered less safe to puncture the auricles; but the interesting paper of Dr. Benj. F. Westbrook, just published in the *Medical Record* for December 23, 1882, seems to show that our fears are as unfounded as were those of our predecessors in regard to ventricular puncture. It is, in truth, to call

attention to his case of harmless *intentional* cardicentesis and to his researches in the surgical anatomy of the operation, that I have been led to refer to the corroborative evidence of the cases mentioned above.

I have with much satisfaction, as have many others, done venesection at the bend of the arm for the temporary relief of the distressing symptoms of dilated heart, and for the dyspnœa due to the pulmonary engorgement of acute pneumonia. If, however, a few *drachms* of blood drawn directly from the heart give the relief that could only be afforded by taking a similar number of *ounces* from the veins of the arm, it seems proper to adopt the former measure. The subsequent circulatory depression from anemia would undoubtedly be less than after the latter operation.

It is manifestly necessary, however, to determine that cardicentesis is innocuous before it can take the place of venesection. The above-mentioned cases and Dr. Westbrook's experience tend to show that such is the fact.

Dr. Westbrook believes that the proper place to perform the operation is in the third costal interspace close to the *right* edge of the sternum. This situation enables the operator to tap the right auricle without injuring the right internal mammary vessels, and with little danger of striking the tricuspid valve. My own preference would be to perforate the ventricle of the right heart by introducing the needle through the fourth interspace, about one and a half or two inches to the *left* of the median line of the sternum. Dr. Westbrook's opinion, however, is entitled to more deference than mine, because he has studied the subject with special reference to cardicen-

tesis, while my special investigations have been limited to the consideration of pericardicentesis.

Further experimentation in heart-puncture for the relief of cardiac distension and pulmonary engorgement is requisite, but it is probable that it will soon become a well recognized surgical procedure in selected cases. Pericardicentesis has already taken that position, and there is no reason to believe that cardiac surgery will stop its march with the demonstration that the pericardium can be treated as the pleura.

In October, 1881, I read a paper before the Anatomical and Surgical Society of Brooklyn (*The Surgery of the Pericardium; Annals of Anatomy and Surgery*, December, 1881), in which I advised resection of costal cartilage and incision of the pericardium for removal of foreign bodies in the pericardial sac; and at the same time said: "The time may possibly come when wounds of the heart itself will be treated by pericardial incision, to allow extraction of clots, and perhaps to suture the cardiac muscle."

It seems as if this time had now almost arrived, for Dr. Block has not only expressed a belief that death can be averted in many cases of heart-wounds by simple incision of the pericardium to allow escape or extraction of the clots which cause pressure and death, but has also undertaken to demonstrate by vivisectal experiments that suture of the heart is a simple operation and requires but three or four minutes (*Amer. Journal of the Med. Sciences*, January, 1883, p. 276; from *Journal de Méd. de Paris*. Oct. 28, 1882; from *Gaz. Méd. de Strassburg*, Oct. 18, 1882). He finds that opening of the right and left ventricles, and entire compression of the heart for the

application of sutures, can be supported by rabbits for several minutes. During suturing he seizes the apex of the heart and draws the organ forward until the traction prevents the escape of blood from the wound. Sutures are then introduced, or the orifice closed by ligation. Even if cardiac pulsation and the respiration stop during this mechanical interference with the heart's movement, death, he asserts, does not necessarily ensue.

These experiments are even more important than the researches spoken of in regard to heart-puncture. I regret that as yet I have not been able to consult Dr. Block's original memoir, but I hope at a future time to do so, and perhaps to be able to report some investigations of my own which I desire to make in the same direction.

OBSERVATIONS ON THE MANAGEMENT OF ENTERIC FEVER ACCORDING TO A PLAN BASED UPON THE SO-CALLED SPECIFIC TREATMENT.

By

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[Read January 3, 1883.]

I DESIRE to lay before the College a plan of managing enteric fever, which I have employed during the past year, and which, tested by such uncertain but not necessarily fallacious means as are available for a limited series of cases, has yielded satisfactory results.

The object of this communication will, I believe, be best attained by first sketching in outline the plan of treatment itself, next by reviewing the considerations which led to its adoption, and finally by a brief study of the cases. This arrangement of the topics will enable us to economize time.

The Plan of Treatment.—The scope of this paper, and the necessity to be brief, debar me from the consideration of the general management of the patient, dietetics, the treatment of complications and sequels and of the prophylaxis, and restrict me, in the main, to the subject of the management by medicinal means. It

is, in fact, this part of the treatment that, superadded to the so-called rational and expectant method in general use in this community, differs from the common practice and constitutes the plan in question.

So soon as the patient is found to have enteric fever, or, in many instances so soon as his symptoms warrant a reasonable suspicion that he is about to develop it, he is put to bed, ordered a diet consisting of milk, animal broths, jelly, and simple custards, in small amounts and at intervals of two or three hours. At night he is given a dose of calomel. This dose varies in amount from 7½ to 10 grains (0.5 to 0.66 gramme) and is repeated every second evening until three or rarely four doses have been administered in the course of the first six or eight days. It is given alone or in connection with sodium bicarbonate. There is commonly a slight increase of diarrhœa, if it be present, without aggravation of the other symptoms, and in some instances the tendency of the temperature at this time to steadily rise, appears to be controlled. If, as is frequently the case, spontaneous diarrhœa has not recurred in the first week, the calomel usually brings about two or three large evacuations on the day following its administration, not more. In either case the tendency to frequent passages in the later stages of the attack is favorably influenced by the repeated administration of this drug during the first week. If the case does not come under observation until after the tenth day, one only, or at most two doses of calomel are given. No further doses of it are, however, given during the course of the attack unless constipation occur. In this event, if the evidences of extensive or deep implication of

the intestinal wall, such as abdominal pain, tenderness, or marked tympany are absent, calomel in $7\frac{1}{2}$-grain (0.5 gramme) doses is given at intervals of three or four days. If there is reason to suspect serious intestinal lesions, the lower bowel may be more safely emptied of its contents every third or fourth day, by enemata of moderate size (8 to 10 fluid-ounces). It is necessary to bear in mind that the gravest lesions of the gut, leading even to hemorrhage and perforation, have occasionally been observed in cases characterized, not only by constipation, but also by an entire absence of pain or tenderness, and very moderate tympany. The danger of salivation from calomel in these doses in enteric fever appears to be slight. In only one case in sixteen were the mercurial fetor and slight swelling of the gums observed.

Excessive diarrhœa has been controlled by the use of opium, either in suppositories, containing 1 grain (0.06 gramme), or by the mouth in quarter-grain (0.016 gramme) doses, often associated with bismuth and given *pro re nata*. It is an invariable rule that the patient be kept in the horizontal position and to the use of the bed-pan and urinal, from the time of the recognition of the disease until defervescence is completed. He is, however, turned upon his side from time to time, and made to maintain that position for twenty or thirty minutes, if necessary, being supported by the nurse.

From the beginning of the attack the following mixture is regularly administered in doses of one, two, or even three drops in a sherry-glassful of ice-water after food, every two or three hours during the day and night.

℞. Tinct. iodinii, f℥ij. 8.00 c. c.
Acid. carbolici liq. f℥j. 4.00 c. c.
M.

Unless some unusual circumstance occur to render a change necessary, this medicine is not suspended until the attack draws to a close. It is well borne by the stomach and excites no repugnance on the part of patients. In one case only has it been necessary to omit the carbolic acid on account of the disgust assumed by its odor.

Partly for the sake of its favorable influence upon the skin and for the sake of cleanliness, partly because of its favorable though slight influence upon the temperature, the patient is to be sponged twice a day with equal parts of aromatic vinegar or alcohol, and cold water. If it is more grateful to him, this sponging may be done with tepid water, the evaporation of an extensive film of water not below the temperature of his body probably being not wholly without a refrigerating tendency.

When the evening axillary temperature reaches 104° F. (40° C.) quinine in massive doses, 24 to 30 grains (1.66 to 2.00 grammes) is given upon a falling temperature. I usually direct 8 to 10 grains to be given in solution at 5, at 5.30, and at 6 A. M. the following morning. Administered thus at the decline of the temperature in its diurnal revolution, these large doses of quinine depress it from 2.5° to 3.5° F. (1.4° to 1.8° C.). After the lapse of forty-eight to seventy-two hours, if necessary the dose may be repeated. If these doses be rejected by the stomach—an unusual circumstance—half the quantity of quinine may be administered hypodermically. For this purpose

a citric acid solution is to be preferred. Since the adoption of the plan of treatment under consideration, I have not encountered cases attended with such hyperpyrexia as has rendered attempts to control it by cold baths necessary or even advisable.

The minor nervous symptoms are best held in check by skilful nursing. For the relief of the headache of the first ten days absolute quietude, a dim light, etc., are often sufficient; occasionally the bromides alone or in combination with chloral are required. Later in the course of the disease chloral is unsafe. From the end of the first week the patient cannot be left unattended even for a few minutes, without risk. Persons in whom delirium was only occasional and transient, have in many instances destroyed themselves during the momentary absence of the nurse.

Alcohol is not often indicated prior to the beginning of the third week. It may, however, by reason of the habits of certain patients, be necessary throughout the attack. Although forming no essential part of the treatment, it is commonly administered in varying though usually small amounts towards the close of the sickness. Some patients do well without taking it at all. It is of course administered in accordance with well-understood indications upon the supervention of delirium, ataxic symptoms, and the evidences of failures of the forces of the circulation. The patients are carefully watched well into convalescence, and cautioned against too soon regarding themselves as restored to health.

The dangers of the establishment of a focus of contagion are guarded against by the systematic.

thorough disinfection of the stools immediately after they are voided.

The considerations which led me to adopt the plan of treatment indicated in the foregoing sketch, are:

1. A feeling of dissatisfaction regarding the expectant method of treating enteric fever. This feeling, vague at first, grew more definite and stronger with increasing clinical opportunities, and a fuller knowledge of the natural history of the disease, until it became a motive, impelling me to cast about for some different and more satisfactory plan. This feeling has been, during the past decade, a very general one in the profession in all parts of the world, as is attested by an almost endless succession of journal articles setting forth new plans of treatment, and the use of new drugs in the management of this, the most common and most important of the acute infectious diseases of the present epoch in medical history. Most of the plans thus suggested have led to disappointment when tested by the fuller observations of the profession; many of them have failed to attract general attention, and some few are still *sub judice*. Their number and diversity bear witness to a widespread distrust of the once well-established expectant treatment. This distrust is, however, based upon something more tangible than a mere feeling of dissatisfaction. The statistics of all observers whose cases have been sufficiently numerous to be trustworthy, show enteric fever to be, when treated by the expectant plan, a disease of high death-rate.

The percentage of fatal cases rarely falls below 15 per cent., and often exceeds 25 per cent., according to the hospital records of this country, Great Britain,

and Continental Europe. Jaccoud, with a collection of 60,000 cases, observed a mortality of 20 per cent.; Murchison, in 27,051 cases, 17.45 per cent.; Liebermeister, in 1718 cases, at Basle, under an expectant plan, records 27.3 per cent. of deaths. But turning from broad generalizations to personal experience, who is there here that, many times elated by the happy issue of mild or average cases treated by the expectant plan, has not realized the sense of utter powerlessness attending it when he has stood face to face with cases in which *to do* rather than *to wait*, has been necessary to save life.

2. Enteric fever is the very type of the general diseases, of affections *totius substantiæ*. The tissues are universally implicated in the morbid processes; no function of the body wholly escapes perturbation For this reason plans of treatment suggested by the prominence of certain groups of symptoms, or by the known lesions of particular organs, even though of undoubted benefit as far as they go, are in theory unsatisfactory because they are directed in effect against conspicuous manifestations of the cause of the sickness rather than against the cause itself.

Whilst in actual practice the treatment by turpentine, by alcohol, by opium with lead, or the silver nitrate, or by agents capable of controlling the febrile movement, as quinine, digitalis, salicin, and the salicylates, even the cold-water treatment itself, although at times and in the hands of certain clinicians showing favorable results—all these have failed of general acceptance on the part of the profession.

3. The general character of the disease, the specific nature of its cause, the unsatisfactory results alike of

an expectant and of a symptomatic plan of treatment, or rather of the two combined, have united to render the idea of a specific treatment, a true cure for enteric fever, a most attractive one, to stimulate thoughtful observers to renew again and again the disappointing search for it. To this idea may be traced the treatment by the mineral acids, by chlorine-water, by carbolic acid, by quinine alone, by quinine and digitalis, by iodine, by the potassium iodide, by calomel.

4. Not only is the conception of a specific treatment for specific diseases a most attractive one, and the attainment of such a treatment for enteric fever brought within the bounds of a reasonable hope by the analogy of syphilis and the malarial diseases, but the search after it with due caution and judgment has also the warrant of the very highest medical authority.

Passing by some earlier names I refer to Da Costa, who has said: "It would be as illogical as absurd to suppose that we shall never possess the coveted means really to cure the continued fevers. Doubtless to the physicians of the time of Charles V. the radical and specific treatment of the malarial fevers appeared as hopeless and remote as the radical and specific treatment of the continued fevers appears to the scientific inquirer of our day."

I refer also to Liebermeister, who, treating about 800 cases, part with calomel, part with iodine, had with the former drug a mortality of only 11.7 per cent., with the latter of 14.6 per cent., against 18.3 per cent. for cases treated without those remedies but in other respects upon a similar plan.

Bartholow has also spoken in favorable terms of the treatment by iodine in combination with carbolic acid.

The treatment adopted is thus seen to consist of the use of the two remedies that are proved to exert a favorable influence upon the disease, iodine and calomel, with the addition of carbolic acid in minute amounts. I am aware that no positive conclusions as to the efficacy of particular plans of treatment can be deduced from a limited series of cases. I am also aware that few acute diseases show greater variations in intensity and in the percentage of mortality at different periods, and under different circumstances, than enteric fever. Nevertheless I have ventured to occupy your attention with this subject to-night because the results of the treatment encourage me to hope that its discussion in this way will lead to its trial on a more extended scale. That it amounts to a specific treatment in the narrow sense is not affirmed. It is tentative, provisional, but it is nevertheless to be regarded as a contribution to the subject of the specific treatment of enteric fever.

The *total number of cases treated* by this plan is sixteen; all recovered, one being now in the second week of convalescence.

Of these, eight were severe, the temperature reaching or exceeding 104° F. (40° C.).

Of these eight severe cases, one was characterized by uncontollable vomiting in the third week. The patient retained no food taken by the mouth for five consecutive days.

One case was very irregular in its course, and was complicated by an obscure abdominal abscess which

discharged by the bowel. The temperature in this case on two occasions attained 105° F. (40.5° C.). This case presented the characteristic eruption of enteric fever.

A third case was prolonged by a severe relapse.

Of the eight cases in which the observed temperature did not at any time attain 104° F. (40° C.), and which were therefore looked upon as medium or mild cases, one was complicated by crural phlebitis, and another by the occurrence of intestinal hemorrhage.

The average duration of the eight severe cases was about 31 days; that of the eight mild and medium cases was about 25 days.

Of the whole number ten were treated in hospital, six in private practice. All from the time of their coming under observation were under my personal care.

In two cases the special plan of treatment was abandoned about the beginning of the third week on account of the supervention of unusual symptoms of great gravity. These related respectively to gastric irritability and an obscure abdominal abscess.

These sixteen cases are unfortunately not a consecutive series. During the year in which I have had the opportunity of observing them, two other cases of enteric fever have occurred in my hospital practice in which this plan of treatment was not employed. One was that of a man suffering from rheumatism, who, after a stay of several weeks in the wards, and in a bed near that occupied by a patient very ill of enteric fever, was observed to be febrile, and to have the typhoid eruption. This person, previously greatly reduced, was not regarded as a suit-

able subject for a special treatment, the efficacy of which was not yet established in my mind. The other was a man who, with an obscure history of a sickness of many weeks, and a very irregular temperature, developed the typhoid eruption, and within forty-eight hours had general peritonitis. These two fatal cases have, however, no bearing upon the result of the treatment.

In private practice several cases of mild continued fever of long duration were treated upon this plan during the past winter. I believe them to have been anomalous cases of enteric fever, but as the rose spots of that disease were absent, and their departure from the typical disease was wide, I have not included them in the above collection of cases treated. They all recovered.

The result of this plan of treatment has not only been satisfactory in respect of the recovery of all the cases treated, an accidental circumstance not liable to mislead persons familiar with the disease, but it has also been satisfactory in respect of the general course of the attack, and the appearance of the patient. These were in the main, despite the severe type of the disease in several of the cases and despite the occurrence of grave complications, favorable. I make this statement with due regard to the personal equation, and with no willingness to permit the observed fact to differ from the actual fact, for I desire any who may make trial of this plan to be more favorably impressed with the results of it, than they have been impressed with my account of it.

[After the reading of the preceding paper:—]

Dr. GEORGE HAMILTON spoke of the great liability to hypostatic congestion in the latter stage of typhoid fever, and asked whether this dangerous condition would not be more liable to ensue by confining the patient from the first and continuously to the recumbent posture. This was one feature of the plan of treatment recommended some time ago by Dr. Wm. Pepper, in typhoid fever, by which he obtained the unequalled result of 98 per cent. of recoveries. He was not, however, at this time able to recall in detail the method of treatment.

Dr. J. T. ESKRIDGE stated that the treatment to which Dr. Hamilton referred consisted in the administration of nitrate of silver, and was that which had been introduced by the late Dr. J. K. Mitchell some years ago.

Dr. ROBERTS BARTHOLOW said that the plan of treatment of typhoid, advocated in the very interesting and able paper just read, is, as all present probably know, in part, the so-called "specific" method. The administration of calomel in full purgative doses during the first week serves a double purpose: it has an effect on the range of temperature, and it acts on the typhoid germs present and multiplying in the intestinal canal. The use of iodine—usually Lugol's solution—throughout the disease, is also one mode of the specific treatment. By the use of this medicine, it is attempted to prevent the multiplication of germs in the intestine, to check fermentation, and to maintain an antiseptic action in the blood.

Although the existence of typhoid germs has not been proved, it must be regarded as possible. Klein, a few years ago, announced the discovery of the specific organism of typhoid in the affected intestinal glands, but Creighton of Cambridge showed that the supposed germs were produced by the mode of preparation. This *fiasco* threw great discredit on the whole question of germs. Nevertheless, the course of treatment directed against supposed germs—the antiseptic method—has had a most favorable influence on the progress and mortality from typhoid. Whilst the specific plan has been advocated in Germany, the Montpelier school has brought forward carbolic acid as the remedy, and the

success which has attended the use of this remedy has been really remarkable. Quite a different complexion has been put on the statistics of mortality since they began the use of carbolic acid. It is probable that the combination of carbolic acid and iodine gives better results than the use of either singly. According to my observation, this method of treatment diminishes the diarrhœa, lowers the fever, and renders the disease much less violent, consequently lessening the mortality. Dr. Wilson has, therefore, rendered us a real service by drawing attention anew to this plan of medication, and especially by supporting his position with valuable cases and statistics. Besides this use of medicines, Dr. Wilson's treatment contains many valuable suggestions and practical methods, which, no doubt, contribute materially to his success.

Dr. J. M. DA COSTA spoke of the purgative treatment in enteric fever, as that which had been tried in the French Hospitals and for a time sanctioned by Louis. As regards calomel it was partly by its purgative action that it was supposed to be beneficial. In his hands the calomel treatment had not yielded favorable results. He had found carbolic acid useful in controlling diarrhœa and in lowering the temperature. He had also employed thymol in one-half to one grain doses. He suggested the use of this remedy in the place of carbolic acid, as more acceptable to the stomach.

Dr. WILSON, in reply to the question of Dr. Hamilton, said that he considered it necessary to frequently change the position of the patient to prevent pulmonary hypostasis. He had intended to emphasize this point in his paper. He called attention to the fact that carbolic acid and like drugs probably exert a favorable influence upon the course of enteric fever by their power to stay the rapid decomposition of the intestinal contents, which, for lack of the antiseptic influence of the intestinal juices, the bile, etc., all of which are changed, is a secondary cause of irritation, diarrhœa, and tympany. Calomel also, he thought, probably exerted an indirect beneficial influence in the same direction.

ARSENICAL PARALYSIS.

By

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[Read February 7, 1883.]

On November 2, 1882, at Norristown, Pennsylvania, occurred a series of cases of arsenical poisoning almost without parallel. The poisoning was brought about chiefly through eating pumpkin-pie, which it was subsequently proved contained a large amount of arsenious acid. The story of the tragedy does not come within my province in the present paper.

On the 24th of December, 1882, I saw one of these cases at Norristown with Dr. E. M. Corson, the physician in attendance. On the 3d of January, 1883, he was brought to the Philadelphia *Orthopædic Hospital and Infirmary for Nervous Diseases*, to be under the care of Dr. S. Weir Mitchell. I am indebted to Dr. Mitchell for great courtesy in affording me the opportunity of thoroughly studying the case at the hospital, and now of presenting it to the College.

Dr. G. B. Massey, Electro-Therapeutist and Assistant Physician to the hospital, carefully studied with me the electrical reactions. The results of our

examinations will be given at the proper place. Dr. Browning, Resident Physician, also rendered assistance in investigating and recording the case.

H. G., æt. 24, single, a lawyer, prior to Thursday, November 2, 1882, was in good health. He was taken sick immediately after rising from the dinner-table, having eaten freely of the pie which was subsequently found to contain the poison. He had an attack of vomiting which lasted a few minutes only. He had several similar spells of vomiting during the afternoon, and from Thursday night until Saturday morning the vomiting was almost continuous. It then began to subside, ceasing entirely Monday afternoon, except that at 2 o'clock Tuesday morning he vomited a dark grumous mass. Just before vomiting this mass he had a sensation of constriction or contraction in the muscles of the chest and throat, and the facial muscles were much contorted. He was not purged at any time; in fact, his bowels were not opened from Thursday, November 2d, until Wednesday, November 8th. During the whole time that the vomiting persisted he had but little pain, scarcely more than would be accounted for by the retching and vomiting. Prostration was very great from the first. Tuesday night, November 7th, he attempted to get out of bed, but fainted, and remained for some time in a semi-conscious state. About this period he began to have marked fever.

On Wednesday, November 8th, and therefore six days after taking the poison, the patient noticed for the first time a sensation of aching and numbness chiefly about the knees. The numbness continued, and, in a few days, extended towards his feet. He still, however, had fair use of his legs, although, of course, they were extremely weak. Three days after the appearance of the numbness in his lower limbs the same sensation began in the fingers of both hands, and soon extended to the wrists, beyond which it never passed.

His brain remained unaffected.

His legs below the knees were now almost completely

paralyzed, and there was some loss of power below the elbows.

His face was considerably puffed and swollen.

His general condition and his special symptoms remained as just recorded, without any change worthy of note, until December 1st, four weeks after the ingestion of the arsenic, when he began to suffer great pain. The pains began in the knees, and speedily invaded the legs below the knees and the feet, progressing in the same course as the numbness had previously taken. Aching was always present, but frequently the pains were boring, tearing, or lancinating. They were accompanied by a sensation like that produced by a strong faradic current. Two days after the coming on of the pain in the legs, the fingers and hands also became the seat of aching. In one week the pains began slowly to abate; but throughout December, and, indeed, up to the present time, he has had more or less pain, varying very much in character. By the middle of December, the numbness and aching, which had previously been below the knees, had extended above them a distance of several inches. His lower extremities felt as if encased in a cylinder as high as the limits of the numbness. The symptoms in his upper extremities did not change noticeably. The loss of power in the thighs increased with the spreading upwards of the sensory perversion.

I will now give the results of my examinations into the condition of the patient. He was first examined by me December 24, 1882, but the notes here given are from examinations made between January 10th and January 17th, 1883, nearly two months and a half after the poisoning.

He presented no brain symptoms, and no disturbances of the special senses of sight, hearing, taste, or smell. He slept fairly well until midnight, and was then usually restless and unable to get into a comfortable position.

He was much emaciated. Wasting of the limbs was extreme. The following measurements were taken:—

Circumference of	right thigh	11 inches.
"	" left "	$10\frac{3}{4}$ "
"	" right calf	$8\frac{1}{4}$ "
"	" left "	$8\frac{1}{4}$ "
"	" right arm	7 "
"	" left "	7 "
"	" right forearm . . .	$6\frac{1}{2}$ "
"	" left " . . .	$6\frac{1}{4}$ "

Paralysis below the elbows was marked, but not complete. The extensors and supinators were most decidedly affected. The fingers could only be flexed about one-half. Movements of the thumbs and the small movements of the fingers were impaired. The loss of power was slightly greater in the right limb than the left. The following were the registerings of the dynamometer:—

Right hand	35
Left "	35

At both elbows were marked contractures at about right angles. The angles could be reduced to about 160°, but any attempt to carry the straightening further caused pain in the flexor tendons.

Both legs were paralyzed completely below the knees. All movements of the toes and feet were abolished absolutely.

The legs in their entirety showed a tendency to rotate outwards, the feet, however, assuming the equino-varus position. Contractures were not present at the knees, but at times the limbs would assume a semiflexed position, these acts of flexure being ac-

companied by cramp-pains in the flexor muscles of the thighs. He had these jerkings both in the legs and arms not infrequently.

The bowels were very torpid, requiring cathartics. Some dribbling of the urine occurred for a few days in the early part of January, and then passed away. For a few days, also, he had some pain, which he referred to the lower part of the urethra, just as the discharge of urine was completed. At the time of examination, January 17th, the urine was passed slowly, but without pain.

The urine showed an excess of phosphates; but neither albumen nor sugar was present.

Farado-contractility was abolished in all muscles below both knees. Above the knees, the extensor and flexor groups and the sartorius were examined, and the faradic reaction was found to be greatly diminished, but not wholly absent. The response was better to nerve than to direct muscular applications.

The muscles below the knees would not respond to weak galvanic currents. To currents of medium strength they responded, but not normally.

The reactions were those of degeneration. Anodal closing gave the strongest reaction; cathodal closure came next. Slight contractions followed both anodal and cathodal opening. The contractions were at first sluggish, though vigorous, increasing after a few trials, and then quickly exhausting. The reactions expressed in the German formula were—

AnSZ″;
KaSZ′;
AnOZ;
KaOz.

In both upper extremities farado-contractility was decreased, but not lost; the diminution was much greater below than above the elbows. Below the elbows the faradic excitability was rapidly exhausted.

To the galvanic current the reactions of degeneration were present, but not so decidedly as in the legs. Anodal closing gave stronger reaction than cathodal. With moderately strong currents tetany was produced at the anode.

Both patellar reflexes were abolished.

The cremaster-reflex was studied, and presented some interesting points. The retraction of the testicle, known as the cremaster-reflex, which has been thoroughly discussed by Dr. S. Weir Mitchell (*Journal of Nervous and Mental Disease*, October, 1879), can usually be awakened by irritation of a certain definite region of the thigh extending from the groin nearly to the knee. In young lads, as Dr. Mitchell has shown, this reflex is easily caused by touching or pinching the whole thigh, with the exception of a band of skin which nearly always may be represented as forming the postero-lateral third of the circumference of the thigh. Sometimes, however, the cremaster-reflex region is far less and sometimes far more extensive. It rarely extends below the knees, although, as stated by Dr. Mitchell, it may in the healthy boy include a large part of the calf of the leg. In adults the excitor region is often much restricted, and it may even be absent.

Gentle irritation of the skin of the inner aspect of the right thigh and leg of the patient, as far down as the malleolus, caused very vigorous retraction of the right testicle. Sometimes, but not usually, both tes-

ticles were retracted. Similar irritation of the left thigh and leg led to movement of the left testicle, which was marked, but not as vigorous as that exhibited by the right from irritation of the right limb. Now and then, in making this test, the unilateral movement of the left testicle, from irritation of the left thigh and leg, was followed a moment later by an imperfect retraction of the testicle of the opposite side. A similar effect was not produced in any of my examinations by irritation applied to the right limb; neither did excitation of one side cause motion in the other side only.

Dr. Mitchell says: "As a rule, which has infrequent exceptions, irritation of one side produces unilateral movement of the testicle of the same side. There are two forms of violation of this law. In the first, irritation of one thigh causes motion of the testicle of the same side, and also, a moment later, less complete action of the testicle of the opposite side. In the other case, touching or pinching certain parts of the inner, and usually of the lower half of the thigh, causes reflex cremaster motion on the other side only; while like irritation in other parts higher up gives rise only to unilateral activity on the same side."

On admission the surface temperature of each calf was 95° F. He usually complained of his legs feeling to him unduly warm.

Late in November transverse white bands were observed across the finger nails about two lines from their posterior limits. The nails were not furrowed, but simply showed white markings. As the nails have slowly grown these lines have remained.

The fingers and forearms were hyperæsthetic, but

at the same time the patient could not determine with any accuracy as to one or two points on testing him with the æsthesiometer. A similar condition, but more marked, was present in the feet, legs, and as high as the middle of the thighs. The muscles were very sensitive.

Applications of hot and cold water were discriminated readily.

I will give the record of pulse, respiration, and temperature, for two weeks from January 9th to 23d.

	Pulse.		Respiration.		Temperature.	
	Morning.	Evening.	Morning.	Evening.	Morning.	Evening.
Jan. 9		107		26		99° F.
" 10	134	140	24	24	98.4° F.	99.2
" 11	134	132	24	24	98.6	98.6
" 12	136	132	24	24	98.8	99.1
" 13	140	128	28	24	98.6	98.8
" 14	140	138	24	28	99.8	98.8
" 15		140		24		98.4
" 16	136	128	24	24	99	98.8
" 17	148	128	24	28	98.8	99.6
" 18	136	124	24	24	99	99.8
" 19	132	116	24	20	98.4	98.6
" 20	124	116	24	24	98.6	98.6
" 21	130	116	24	24	97.8	98.6
" 22	128	120	24	24	98	98.8
" 23	124	136	24	25	99	99.4

The pulse, therefore, during the time of these observations, ranged between 107 and 148, and was nearly always more rapid in the morning than in the evening.

The respirations ranged between 20 and 24, standing usually at about 24.

The temperature ranged between 97.8° F. and 99.8° F., but commonly was not much either way from the normal.

On admission to the hospital the following treatment was instituted by Dr. Mitchell: Applications of ice and hot water alternately were made three times daily for ten minutes at a time to his arms and legs from the elbows and knees downward. Surface massage with cocoa-nut oil was used once daily. Ice-bags were applied to the spine for one to two hours twice daily. One grain of the extract of ergot of the new United States Pharmacopœia was given every two hours, and this was rapidly increased until thirty grains daily were administered. After continuing the use of the ergot for a week the patient's stomach became disordered, and tincture of belladonna in doses of five drops every three hours was substituted. Fifteen grains of chloral were administered occasionally, and sulphate of morphia, at first $\frac{1}{25}$th grain, and eventually increased to $\frac{1}{16}$th grain, was ordered, to relieve pain when necessary.

He was placed on the ordinary full diet of the hospital, with the addition of milk three times daily and beef-tea twice daily.

I made an examination of the patient to note the effects of treatment to-day (February 7th). He has improved steadily day by day. His general strength has increased. He has regained almost entirely the use of the muscles above the knees. He has also much better use of his forearms and hands, particularly the latter, being now able to pick up small objects. The "wrist-drop" has improved greatly. He has much less pain, aching, and numbness below the knees; the legs below the knees, however, still remain paralyzed, but are not so completely helpless. He has every appearance of progressing steadily to recovery.

Having presented this case as fully as possible, I will give in a few words all the information I have been able to obtain as to paralysis and other nervous symptoms shown by the other victims of the poisoning.

Six others altogether, besides our patient, were poisoned. One of these was a little boy, 4 years old, I. S., to whom a piece of the fatal pie was given as a reward for going on an errand. He died within ten hours, and I have no knowledge of observations as to paralysis or other manifestations of involvement of the nervous system. Probably his death occurred too soon to allow any such observation to be made.

M. S., a sister of the little boy, ate a very little of the pie, and suffered to some extent, but not seriously.

C. H. G., the father of the patient, died November 8th, six days after the ingestion of the arsenic. Besides severe gastro-intestinal symptoms, he suffered with pain in his head, back, and limbs, was delirious for some hours, and was almost completely paralyzed.

Mrs. G., mother of the patient, ate a little of the pie November 2d, and had an attack of vomiting. On the 4th she ate a piece of custard, which was also found to contain arsenic, and was attacked with vomiting. A few days later weakness of the legs, with aching and numbness, came on, and the right foot and leg became swollen and inflamed. She gradually recovered.

Mrs. V. ate a mouthful or two of the pie and custard containing the arsenic, and suffered with vomiting, etc., for three days. She has since had paresis and paræsthesia of the legs.

Mrs. F., who ate freely of the poisoned food, suffered severely from gastro-intestinal symptoms. She is now under the professional care of Dr. H. N. Umstead, of Yerkes P. O., Montgomery Co., Pa., who has courteously written to me about her condition, and the substance of whose communication I will give. Dr. Umstead states that Mrs. F. has been

paralyzed from the elbows to the ends of her fingers and from the knees to the toes. She complained of numbness and coldness in the limbs, and a feeling as if a cord was tied tightly around the waist. She had extreme pain in the paralyzed extremities. She has greatly improved, is riding out daily, can stand without aid, and can even walk a little with assistance. She still has some pain in the hands and in the soles of the feet, but they are not tender to the touch. She has some anæsthesia of the hands and feet, especially of the latter. She begins to enjoy her food, for which at first she had great loathing. Her bowels are moved once, and she urinates twice daily, but has not quite the natural sensation when the bowel or bladder is evacuated. Dr. Umstead states that Mrs. F. and Mrs. V., whom he also treated, make five cases of arsenical paralysis which have fallen under his care during his professional career.

A careful analysis of the history and symptomatology of the case detailed must compel me to conclude that in well-marked arsenical paralysis we have to deal with a diffused myelitis, decided motor, trophic and sensory bilateral phenomena being present.

With the assistance of Dr. J. H. Lloyd, one of the staff of the Nervous Dispensary of the University Hospital, I began the preparation of a *résumé* of the literature of arsenical paralysis, when the *Journal of Nervous and Mental Disease* for October, 1882, containing an admirable article on the subject by Professor E. C. Séguin, came to hand. In this paper the literature of the subject is given with considerable fullness, and to it I would refer those interested.

Beginning with Abano, who flourished as early as the 13th century, the authorities quoted or referred to by Professor Séguin are Forestus, Zacchias, Hah-

nemann, Thilenius, Brodie, Orfila, Christison, Graves, Huss, Leroy d'Étiolles, Imbert-Gourbeyre, Smoler, Jaccoud, Seeligmüller, Popow, Rosenthal, Romberg, Erb, Hammond, and Da Costa.

I will refer here only to the observations of Christison and Popow. Christison describes two classes of cases of arsenical poisoning in which the victims die early without paralysis, and a third class of what he terms subacute cases, with moderate gastro-intestinal inflammation. "In the later stage these cases are apt to show marked nervous symptoms: coma, epileptoid attacks, mania, tetanus, hysterical seizures, partial paralysis resembling lead, paralysis in affecting the extremities; contractures may exist."

In 1881, Popow, of St. Petersburg, published an essay upon the pathological anatomy of arsenical paralysis as produced artificially in animals. The work of Popow was carried on under the guidance of the distinguished neuralogist and microscopist, Professor Mierzeyewski, and Séguin considers his essay as in many respects the most important contribution yet made to the subject. Popow concludes that arsenic, even in a few hours after its ingestion, may cause acute central myelitis or acute poliomyelitis; that in chronic cases pathological changes are found in the white as well as in the gray substance, constituting a diffused myelitis; and that the peripheral nerves remain normal, even three months after intoxication. Séguin gives condensed accounts of a few of the cases reported in the literature of the subject, and also reports three cases of his own, all would-be suicides with Paris green. His conclusions are practically the same as those of Popow. According

to Séguin, whether the myelitis is strictly arsenical, *i. e.*, caused by the direct effect of the arsenic on the tissue of the spinal cord, or whether it is produced (as are many forms of myelitis) by the irritation of peripheral nerves (cutaneous, intestinal, and gastric nerve-endings), is a question which cannot at present be definitely solved, but which presents an interesting field for future research and speculation.

Dr. Lloyd has collected the following references to authorities and cases in addition to those cited by Séguin:—

Beck (*Elements of Medical Jurisprudence*, sixth edition, vol. ii. 1838) gives three classes or varieties of arsenical poisoning. In the 3d variety there is, first the inflammatory action, then, when this recedes, comes the second stage, that of nervous involvement. The nervous symptoms vary "from coma to an imperfect palsy of the arms and legs, and between these extremes are observed epileptic fits or tetanus."

Taylor (*On Poisons, etc.*, 1848) gives several cases where the symptoms of *narcotism* (or general paralysis of the nervous system) were marked.

A man swallowed, by accident, some arsenic early in the morning. He went to work for several hours afterwards; and was then gradually observed to sink into a drowsy state, and died that night with no complaint of pain.

A child, aged $2\frac{1}{2}$ years, died *narcotized* two hours after taking the poison.

Wharton and Stillé (*A Treatise on Medical Jurisprudence*, second and revised edition, 1860) make mere mention of palsy as a symptom which is apt to occur late in the case.

Taylor (*A Manual of Medical Jurisprudence*, seventh American edition, 1873) speaks of local paralyses, preceded by numbness or tingling in the fingers and toes, as common consequences of chronic arsenical poisoning.

According to Stillé (*Therapeutics and Materia Medica*, 4th ed., vol. ii. 1874, p. 816) arsenical paralysis most frequently affects the lower limbs first, extending gradually to the arms; but it is more permanent in the legs, continuing for months or even years. It is accompanied with cramps, spasmodic movements, numbness, and formication. The cutaneous sensibility is impaired, and the patient generally complains of coldness in the parts affected.

H. C. Wood (*A Treatise on Therapeutics, etc.*, second edition, 1876) speaks of paralysis, which follows non-fatal cases, and affects preferably the lower extremities, commencing and remaining longest in them; does not select the exterior muscles, and is almost always accompanied by anæsthesia, or at least by numbness and formication, and by coldness of the extremities. He quotes experiments on frogs; mostly from Sklarek.

Ringer (*Hand-Book of Therapeutics*, ninth edition, 1883) refers to his experiments on frogs. He found paralysis of sensation, reflex action, and voluntary motion. He believes that the paralyzing action is exerted on the cord first, then on the nerves, and last on the muscles. The difference may be noted between Ringer and Séguin, that Ringer does not refer all pathological changes to the cord. He regards the arsenic as a "protoplasmic poison," affecting all tissues. He says that frogs are sometimes only appa-

rently paralyzed, *i. e.*, sensation is lost, and hence there is no response to external irritants, but if laid on their back, they turn themselves over.

The Index-Catalogue of the Surgeon-General's Office gives the titles of 43 books on the Physiological and Therapeutic Effects of Arsenic, but nothing special on its paralyzing effects.

Gibb ("Neuralgia and Paraplegia, supposed to be due to long-continued use of arsenic, etc.," *Trans. Path. Soc. Lond.*, ix. p. 442) records the case of a lady who had taken arsenic, mostly Fowler's solution, for many years for a skin affection. She had attacks of acute neuralgia in groins, shoulders, and sides. These pains were considered due to arsenic by Sir Jas. Clark, Dr. Robert Lee, and Dr. Copland, who all saw the case. Afterwards she lost all power over her lower limbs, which felt numb, although sensibility remained perfect. This retention of sensibility is at variance with other observers and experimenters. After death the abdominal and thoracic glands were found enlarged, and traces of arsenic were found in liver and lumbar vertebræ, although the drug had not been taken for more than seven months before death.

Colton ("Arsenical Paralysis," *N. Y. Journ. of Med.*, September, 1850, p. 177–78) mentions the case of a patient who swallowed, accidentally, some arsenic, and was admitted to the hospital under Dr. Colton's care. The primary effects of the poison had been successfully combated with proper remedies. Seven days afterwards, when feeling quite well, he was attacked with violent cramps in index finger of right hand, spreading to other fingers, then to other hand, and finally to the feet. The pain in hands subsided as the

feet became affected. The cramps lasted 30 minutes. He then fell into sound sleep, it being night, but in the morning he found to his surprise that he had lost the use of the affected parts. This paralysis had continued unchanged for five months. There was also a feeling of heat and numbness in the arms from the fingers to a little below the elbows, and in the legs from the toes to a little below the knees. Lancinating pains also occurred in those parts daily from 5 P. M. to midnight. He improved slowly under the use of quinia, strychnia, and electricity.

McCready ("Death from External Application of Arsenic," *Am. Journ. Med. Sci.*, July, 1851, p. 259) relates that a woman rubbed white arsenic mixed with gin on the head of her child suffering with favus. The child died in less than 48 hours with its legs completely paralyzed.

Maclagan ("On the Arsenic Eaters of Styria," *Ed. Med. Journ.*, 1864, p. 200) visited Styria in the year 1864, and had personal interviews with two "arsenikophagites;" one of whom ate in his presence nearly 5 grs. of arsenious acid, and the other nearly 6 grs. The urine of both of these men was carefully bottled and taken back to Great Britain, where a chemical examination revealed arsenic. The physiological effects on these toxicophagi are described as being only tonic and stimulant, especially improving the wind and increasing sexual desire. Maclagan's evidence is strong, yet perhaps not such as would be received in a court of justice, as the men were not kept continuously under observation. The point of chief interest is that he says nothing about paraplegia or any acute

or chronic poisoning symptoms among these arsenic eaters.

Dr. Lloyd has called my attention to a case, not before reported, of suicide with arsenic, which happened some years ago in Bucks County, and was under the care of the late Dr. Hendrie. Anæsthesia and paralysis were so marked that the man declared that his legs were cut off, and died in that belief.

[After the reading of the preceding paper:—]

Dr. S. Weir Mitchell asked if the urine had been examined with care in the early stages of the case. Of late there had been no evidence of trouble, and if at a former period there was albumen, it was no longer present.

Perhaps it was not known to all the Fellows that arsenic, in medicinal doses, was in rare cases, as Dr. M. pointed out many years ago, the cause of more or less albuminuria.

As concerned diagnosis, Dr. M. had always looked on these grave forms of paralysis from arsenic as due to myelitis, and saw much in this case to support and nothing to oppose this opinion. Among the symptoms on which the author of the paper had dwelt least were the frequent twitches of the limbs, especially in sleep, and the intense general tenderness of the muscles, which disappeared readily under the use of massage.

The pearly tinted band on the nails, about one line wide, had not the slightest indentation, and was unlike anything in the way of an indication of arrest of nail growth which has ever come to Dr. M.'s attention.

Dr. Roberts Bartholow said that these forms of arsenical poisoning, affecting the nervous system, as described in the very interesting paper by Dr. Mills, present many remarkable features. It has long been known that there are cases in which profound depression of the nervous centres, coma and insensibility, have been caused by large doses of arsenic, without any local irritation—without gastro-intestinal inflammation. On the other hand, Virchow informs us, that there are cases of acute arsenical poisoning which cannot be differentiated either in respect to the symptoms observed during life or in the morbid anatomy from the algid stage of cholera. The author of the paper did not refer to the fatty degeneration of the intima of the vessels, or to the same change occurring in the epithelial structures of various organs, but he gave an account of the other changes, all of which show the profound alterations to which the tissues of the body in general are subjected, and which tend to prove the correctness of Ringer's view, that arsenic is a protoplasmic poison, and as such leaves no part of the organism untouched. There is doubtless a

community of actions amongst the poisonous metals, and all affect the system to a less or greater extent in the same way. The metals are so largely employed in trades and in domestic life in our day, that many cases of obscure nervous diseases may have their origin in this way. In respect to the treatment pursued, he would have directed more attention to securing elimination of the poison. However, on this point it must be admitted that the time during which elimination can be effected is rather short. The chemists tell us that if, in a fatal case of arsenical poisoning, the patient lives a week after the poison has been swallowed, its detection may be impossible, so rapidly is it eliminated.

In reply to Dr. Bartholow, Dr. Mitchell said the time for attempts at elimination had passed, as two months had elapsed between the poisoning and the patient's admission to the hospital.

Dr. S. W. Gross asked whether any observations had been made with regard to the genital functions in the case reported.

Dr. J. T. Eskridge said that the lecturer had not referred to changes in the blood in acute arsenical poisoning. Brodie, quoted in Stillé's work on Materia Medica and Therapeutics, observed a fluid condition of the blood in animals poisoned by arsenic. He called attention to the fact, because it was another proof of the profound devitalizing influences of the drug when taken in toxic doses.

Dr. Mills, in reply to the questions which had been asked, stated that there was impairment of the genital functions, but that sexual desire and evidences of sexual power were present. So far as he knew, the urine had not been examined in the early stages of the case. Efforts were made by Dr. Corson, under whose care the patient came, to eliminate the poison by cathartics. When he saw the patient first, the time had passed to derive much benefit from this plan of treatment. Iodide of potassium was administered.

A PARTIAL STUDY OF THE POISON OF HELODERMA SUSPECTUM (COPE)—THE GILA MONSTER.

By

S. WEIR MITCHELL, M. D., and EDWARD T. REICHERT, M. D.

[Read February 7, 1882.]

For some years past it has been known to naturalists that the Gila lizard of Arizona and Sonora was endowed with anterior deciduous grooved teeth, which communicated by ducts with large glands within the angle of the lower jaw. These arrangements naturally suggested a certain power of poisoning, as to which, however, the most conflicting accounts have reached, and continue to reach us from Arizona. In many houses the sluggish creature shown to you was a pet of children, and seems to have been averse to using his weapons of offence. The occasional accidents from his bite were variously explained away; but still, among the Indians and some settlers, he enjoyed an evil reputation. Only within a week we have had two letters from Arizona, the one describing him as "more peaceful and harmless than a young missionary," and the other as being "worse than a whole apothecary shop." Nevertheless, both in France, and of late in London, specimens have bitten and promptly killed small animals.

It is worth while to mention more distinctly some of the evidence for and against the poisoning power of Heloderma. His bad name in Mexico is mentioned by Bocourt and Dumeril, but Sumichrast is more full in his statements.

This curious lizard is, he says, slow and embarrassed in his movements, and hides in the daylight, and especially in dry weather, to emerge at night and in wet seasons. He is said to smell ill, in fact to be of a nauseating odor, and is described as slobbering forth a sticky, whitish saliva when irritated. The natives, says Sumichrast, hold him in the utmost terror, and consider him as more fatal than any serpent. When made to bite a fowl, it died in twelve hours, with bloody fluid exuding from its mouth, the wound being of a purple tint. A cat bitten was very ill, but recovered, remaining thin and weak. The Heloderma horridum sent to London, to Sir John Lubbock, killed a frog in a few minutes, and a guinea-pig in three minutes.

Many years ago Dr. Irwin, U. S. A. (*Amer. Naturalist*, Nov. 1882), experimented in New Mexico with the Gila monster, and concluded it to be harmless, while Mr. Horan, Superintendent of the National Museum, says he himself has been several times bitten without serious results. The following statement of Dr. Shufeldt (*Amer. Naturalist*, Nov. 1882) adds a further difficulty in making up our estimate of the powers of Heloderma. The lizard he speaks of is the one we now exhibit. It was sent to the Smithsonian Institution by A. T. Burr, U. S. A., and is the H. suspectum of Cope.

On the 18th inst., in the company of Professor Gill of the Smithsonian Institution, I examined for the first time Dr. Burr's specimen, then in a cage in the herpetological room. It was in capital health, and at first I handled it with great care, holding it in my left hand, examining special parts with my right. At the close of this examination I was about to return the fellow to his temporary quarters, when my left hand slipped slightly, and the now highly indignant and irritated Heloderma made a dart forward and seized my right thumb in his mouth, inflicting a severe lacerated wound, sinking the teeth in his upper maxilla to the very bone. He loosed his hold immediately and I replaced him in his cage, with far greater haste, perhaps, than I removed him from it.

By suction with my mouth, I drew not a little blood from the wound, but the bleeding soon ceased entirely, to be followed in a few moments by very severe shooting pains up my arm and down the corresponding side. The severity of these pains was so unexpected that, added to the nervous shock already experienced, no doubt, and a rapid swelling of the parts that now set in, they caused me to become so faint as to fall, and Dr. Gill's study was reached with no little difficulty. The action of the skin was greatly increased, and the perspiration flowed profusely. A small quantity of whiskey was administered. This is about a fair statement of the immediate symptoms; the same night the pain allowed of no rest, although the hand was kept in ice and laudanum; but the swelling was confined to this member alone, not passing beyond the wrist. Next morning this was considerably reduced, and further reduction was assisted by the use of a lead-water wash.

In a few days the wound healed kindly, and in all probability will leave no scar; all other symptoms subsided without treatment, beyond the wearing for forty-eight hours so much of a kid glove as covered the parts involved.

After the bite our specimen was dull and sluggish, simu-

lating the torpidity of the venomous serpent after it has inflicted its deadly wound, but it soon resumed its usual action and appearance, crawling in rather an awkard manner about its cage.

The specimen shown has eaten once since we have had him, but the Gila monster is said to live on bird eggs, and to eat daily of like food while in captivity.

The sluggish habits ascribed to Heloderma in general have been noticed in our specimen; but it is clear from Dr. Shufeldt's accident, that, like the habitually inert Crotalidæ, this creature is capable of sudden, and therefore, unexpected agility in attack.

As we shall have sent to us in the spring a number of Helodermas, we shall then be able to complete the study of the poison of these interesting lizards—the only members of the family of lizards as yet known to be poisonous. The subject is, however, too full of interest to delay the publication of our preliminary study, since, as far as it has gone, it is perfectly definite and satisfactory.

The Gila monster inhabits the dry hillsides of Arizona, and is said to reach the length of three feet.

The specimen we exhibit is about fourteen inches long, and from war or accident, had, when he reached us, lost all but two of his teeth, and as yet no new ones have taken their places. Without them he would certainly be as harmless as a rattlesnake deprived of his fangs; and as these teeth are very small and easily removed, their absence may account for

some of the instances in which the lizard has bitten and done no grave harm.

Experiments made in the usual vague way, by allowing the lizard to bite animals, are obviously untrustworthy; so that it was thought best to use the saliva in known quantities. The fluid was obtained by provoking the animal to bite on a saucer-edge—which it was most indisposed to do. When once it had seized the saucer it was hard to pull it away, so powerful was the grip of the lizard's jaws. After a moment, a thin fluid like saliva dripped in small quantities from the lower jaw. It was slightly tinted with blood, due to the violence of the bite, and it had a faint and not unpleasant aromatic odor. The secretion thus collected from the mouth was distinctly alkaline, in contrast to serpent venoms, which are all alike acid.

Experiment I.—About four minims were diluted with one-half cubic centimeter of water, and thrown into the breast muscles of a large, strong pigeon, at 4.23 P. M. In three minutes the pigeon was rocking on its feet, and walking unsteadily. At the same time the respiration became rapid and short, and at the fifth minute feeble. At the sixth minute the bird fell in convulsions, with dilated pupils, and was dead before the end of the seventh minute.

The first contrast to the effects of venom was shown when the wound made by the hypodermic needle was examined. There was not the least trace of local action, such as is so characteristic of the bite of serpents, and especially of the Crotalidæ.

The muscles and nerves responded perfectly to weak induced currents, and to mechanical stimuli.

The heart was arrested in the fullest diastole, and was full

of firm black clots. The intestines looked congested. The spine was not examined.

Experiment II.—In the following experiment a full-grown etherized rabbit was used, and the left carotid being connected with the kymographion, one-sixth of a grain of dry Heloderma venom dissolved in one cc. of distilled water, was injected into the external jugular vein.

Action on the Arterial Pressure.

	Time. min. sec.	Pressure. mm.	Remarks.
Normal,		110	
Injection,	.0		
	.3	100	
	.5	80	
	.10	60	
	.15	70	
	.20	66	
	.30	50	
	1.	50	
	2.	44	
	3.	32	
	4.	26	
	5.	20	
	8.30		
	10.		
	10.30		
	11.10		
	11.30		Convulsive movements.
	12.		The pressure gradually declining to 7 mm., when the animal expired.
	12.30		
	14.30		
	15.		
	16.		
	17.		
	18.		
	19. Death.		

Action on the Pulse.

	Time. min. sec.	Pulse. in 10 sec.	Pulse Curves. mm.	Remarks.
Normal,		57	.7	
Injection,	.0			
	.15	57	1.0	
	.30	54	1.2	
	1.	51	1.0	
	2.	53	.8	
	3.	56	.7	
	4.	61	.6	
	5.	61	.5	
	8.30	56	.4	Convulsive movements.
	10.	47	.5	
	10.30	27	1.6	
	11.10	31	1.0	
	11.30	19	1.2	
	12.00	22	.8	
	12.30	28	.3	
	14.30	63	.2	
	15.	58	.3	
	16.	63	.2	Convulsive movements.
	17.			
	18.			Too feeble to count.
	19.			
	Death.			

Experiment III.—The following experiment was made on a full-grown rabbit in which the pneumogastric nerves were cut, and in which the same dose and method of injection were used; the object being to determine if the above nerves were in any way connected with the changes in the circulation observed in the preceding experiments.

Action on the Arterial Pressure.

	Time. min. sec.	Pressure. mm.	Remarks.
Normal,		80	
Injection,	.0		
	.8	66	
	.15	52	
	.30	56	Convulsive movements.
	.40	60	" "
	.50	42	" " stopped.
	1.	34	
	.10	28	" "
	.30	24	" "
	.35	30	Violent convulsions followed by death in 30 seconds. During these convulsions the canula became detached from the artery.
Death in 1 min. 35 sec.			

Action on the Pulse.

	Time. min. sec.	Pulse. in 10 sec.	Pulse Curves. mm.	Remarks.
Normal,		46	.6	During the last half of
Injection,	.0			the first minute, and
	.15	47	.8	after, the tracing was
	.30	44	1.0	so irregular on account
	1.00	52	.3	of the convulsive
	1.30			movements that the
	1.35	Violent convulsions.		pulse could not accu-
				rately be counted.

The animal died in convulsions with dilated pupils.

The results were identical with those obtained when the pneumogastrics were entire, so that the effect on the heart is direct, and not by inhibition through the pneumogastrics.

The results of the autopsy in both of the above experiments are identical, and may be summed up as follows:—

Autopsy, made immediately after death.—Heart arrested in diastole; heart does not react to induction currents; muscles everywhere respond to electric stimulation; motor nerves intact; cord unirritable,

and will not respond to the strongest current produced by one large gravity cell, with Du Bois Reymond's induction coil; bowels still irritable; peristaltic movements occur spontaneously; the intestines are natural in color, as are all other organs. After five minutes the heart began to contract, and was finally found in a systolic condition. The interior of the organ was full of black clots, especially the auricles, the left ventricle containing but a very small clot.

In order still further to determine the effect on the heart, the following experiments were made:

Experiment IV.—

hrs.	min.	
7.	33	Pithed frog and exposed the heart.
	.52	Heart beats 21 in 30 seconds.
	.52½	Placed a small portion of dried venom of Heloderma on the heart.
	.56	Heart beats 20 in 30 seconds.
8.	05	" " 19 " "
	.18	" " 18 " "
	.30	" " 15 " "
	.43	" " 14 " "
	.55	" " 10 " "
9.	30	" " ceased.

Experiment V.—

hrs.	min.	
8.	05	Took two "cut out" hearts of frogs, and placed them in a normal salt solution in separate vessels, just sufficient liquid being used to cover the hearts. On one heart was placed a small quantity of dried venom.
	.27	The poisoned heart beats more feebly than the other.
	.30	The poisoned heart beats still more feebly than the other, which is yet firm.
	.45	The poisoned heart stopped beating, the other beats firmly.
	.55	The poisoned heart stopped beating, the other beats firmly, but slower.

Experiment VI.—

hrs. min.

3.45 Exposed the hearts of two pithed frogs.

4.00 Placed on one some dried venom.

.30 The poisoned heart beats are decidedly feebler than the other.

.50 The poisoned heart beats more feebly; the unpoisoned heart beats firmly and apparently in a normal manner.

6.00 The poisoned heart beats very feebly and does not fill with blood. The normal heart beats firmly, and fills well with blood at each beat, making a striking contrast with the poisoned heart.

We may conclude that—

The poison of Heloderma causes no local injury.

That it arrests the heart in diastole, and that the organ afterwards contracts slowly—possibly in rapid rigor mortis.

That the cardiac muscle loses its irritability to stimuli at the time it ceases to beat.

That the other muscles and the nerves respond readily to irritants.

That the spinal cord has its power annihilated abruptly, and refuses to respond to the most powerful electrical currents.

This interesting and virulent heart poison contrasts strongly with the venoms of serpents, since they give rise to local hemorrhages, and cause death chiefly through failure of the respiration, and not by the heart, unless given in overwhelming doses.

They lower muscle and nerve reactions, especially those of the respiratory apparatus, but do not, as a rule, cause extreme and abrupt loss of spinal power.

Finally, they give rise to a wide range of second-

ary pathological appearances, which are absent from Heloderma poisoning.

There remains on our minds no doubt as to the fact that the fluid which drips from the mouth of Heloderma when it bites is a very active poison. The present study is, however, limited in range, and we cannot yet feel sure that the fluid in question comes from the glands now presumed from their relation to the teeth to be poisonous.

The briefest examination of the lizard's anatomy makes clear why it has been with reason suspected to be poisonous, and why it poisons with so much difficulty. Unless the teeth are entire, the poison abundant, and the teeth buried in the bitten flesh so as to force it down into contact with the ducts where they open at the crown of the teeth, it is hard to see how even a drop of poison could be forced into the wounds. Yet it is certain that small animals may die from the bite, and this may be due to the extraordinary activity of the poison, and to the lizard's habit of tenaciously holding fast to what it bites, so as to allow time for a certain amount of absorption.

It is plain enough that a lizard as small as the one exhibited would be very unlikely to inflict a wound fatal to man; but it is possible that the larger animal —and it is said to reach a length of three feet—might prove a more efficient poisoner.

We are unwilling to drop the subject without a few words as to the nature of this poison.

The recent researches of Dr. Sternberg and Prof. Gautier have shown that human saliva may kill a rabbit in twenty-four hours (Dr. S.), and a pigeon in a few hours (Prof. G.), if a quantity of saliva have

been concentrated by heat and so used. Professor Gautier thinks the saliva and all venoms owe, at least, a part of their power to normal ptomaines or animal alkaloids, the products of putrefactive processes, and recalls to us the fact that most secretions are measurably poisonous.

The answer to these views we shall have to consider elsewhere, and at length, but it will be sufficient here to say that there is no resemblance between the symptoms caused by the known ptomaines and those produced by any of the venoms. When it was shown that healthy human saliva was competent to kill, it was natural enough to leap to the conclusion that the venoms were merely concentrated salivas. The analogy ends with the fact that both may cause death, but the one may kill in twenty seconds, and the other requires, at the least, many hours, whilst also it seems, as regards saliva, to be, in some degree, a question of the toxic activity of certain individuals, not all being so uncomfortably endowed as Dr. Sternberg himself.

THE HISTORY OF A CASE OF ABDOMINAL CYSTIC TUMOR, WHERE SEVEN YEARS AFTER REMOVAL OF THE TUMOR BY LAPAROTOMY A SECOND OPERATION WAS DEMANDED:

TAPPING THROUGH THE VAGINA RESORTED TO, WITH CONSEQUENT DEATH OF THE PATIENT.

By

WALTER F. ATLEE, M.D.

[Read March 7, 1883.]

Bene facit qui ex erroribus suis exemplum aliis præbet.

In June, 1875, I removed, by laparotomy, in the St. Luke Hospital, Bethlehem, a multilocular cyst from Mrs. DeM——. The history of the case was given by Dr. Stout to the Board of Trustees, and published in their second Annual Report. The whole mass removed at the operation weighed seventy-eight and a half pounds. The lobes and lobules composing it were made up of cysts containing fluids of different densities, colors, and other physical properties. The pedicle appeared to arise from the rectum, at all events, the cyst wall was separated from everything else, except the front of that intestine, and the only ligature used was there applied. The whole vascular supply was derived from meso-rectal vessels. Of course the clamp could not be used. As always, in sewing up the incision in the abdominal walls, I carefully in-

cluded the peritoneum in the sutures, as in this way adhesions of intestine and omentum, with the consequent discomfort and constipation suffered by the patient, are never observed, and, moreover, in case a second ovariotomy should have to be performed, the operation could be performed with much more ease and safety.

This patient recovered from the operation, though somewhat slowly, owing to delay and difficulty in removing the ligature, which was left hanging out of the external wound, and not cut short and left inside the abdomen, as is now generally practised. She remained in perfect health for seven years, but in the summer of 1882 came to my office complaining of considerable discomfort in the pelvis, where I detected, by the touch, the presence of a swelling, caused by a thick, viscid fluid; in other words, there was plainly a second tumor formed there. This continued to enlarge, and in the month of January, the suffering it occasioned was so immediately threatening to the patient's life, that it was necessary to act. Great efforts were made to push the tumor out of the pelvis, but this was not to be accomplished. It remained to operate as before, and remove the mass by laparotomy, or to give relief by emptying the cyst by tapping through the vagina. The latter procedure was preferred, and about three pints of a very thick, dark-colored fluid were drawn off by a Thompson trocar. There was no difficulty about the operation, nor was it attended by any particular pain, nor was there any hemorrhage. The next day the patient felt herself completely relieved, but after that her pulse became quickened, and a general febrile condition

unpreceded by chill, succeeded. There was evident tenderness on pressure over the lower part of the abdomen, but no severe pain independent of this pressure. The stomach became so irritable as to reject everything swallowed. Death took place on the eighth day, under the marked symptoms of depression which accompany pyæmia. It was a death, most plainly, from cyst inflammation.

Notwithstanding the great number of cases of operation for ovarian disease reported nowadays in journals, more or less medical, this case is really one deserving of record, and one of true and special interest to the pathologist and to the surgeon.

In removing the large mass in the first operation, in this case, it was noticed that no pedicle was found. It was separable by the fingers everywhere except from the front of the rectum, where it was necessary to apply a ligature and divide the attachment by scissors. The ovaries, as we all know, are situated in the posterior fold of the broad ligaments, on the sides of the highest part of the uterus, behind the Fallopian tubes and the round ligaments, which separate them from the bladder, and in front of the rectum, from which they are commonly separated by the lowest circumvolutions of the ileum. By a rounded cord, the ligament of the ovary, they are attached internally to the womb, and externally by two folds of the peritoneum to the sides of the pelvis.

As the ovary, in its normal condition, is not pediculated, at first, of course, a diseased ovary has no true pedicle. As it becomes larger, this pedicle is formed from changes in its normal connections with the adjacent womb; it consists of the Fallopian tube often

much elongated, the broad ligament often considerably thickened, the utero-ovarian ligament occasionally hypertrophied to a large fibroid stem, the round ligament, and enlarged bloodvessels. If the tumor rises out of the pelvis, the pedicle will be longer, and if bound down in the pelvis from early adhesions it will be shorter.

In some cases, however, and they are by no means rare ones, when cystic tumors are removed from the abdominal cavity, no such pedicle as the one just described is met with. In the case whose history I have been relating, it was said that there was no distinct pedicle, and that the whole vascular supply came from the vessels of the meso-rectum. It may naturally be supposed that this was owing to the way in which the diseased mass may have been separated from its surroundings, and I therefore call attention to the fact that the same thing has occurred in the practice of an operator such as Spencer Wells. In his *Diseases of the Ovaries*, in relating Case CX., he says that he found no pedicle, and that the tumor derived its vascular supply from the omental and mesenteric vessels.

It seems to me most reasonable to look upon some of the abdominal tumors having these connections, not as enlarged and diseased ovaries, but as changed ovules, which have failed to enter the Fallopian tubes, have slipped in the interstices of the neighboring organs, become attached, received blood, and grown. In women, when the Fallopian tube has become adherent, in place of floating loosely, the ovule is not grasped as it leaves the ovary, and then it is not rare to see ten, fifteen, twenty, or even more small cysts, some as large as a pea, on the parts immediately in

contact with the ovary. (See Sappey, vol. iii. p. 644.)

If one of these misplaced ovules was attached to a spot where plenty of blood was supplied, a large cyst could be formed, having no distinct pedicle, as in the case before us. As is seen in cases of extra-uterine fœtation, a well-made child can be formed in this way from a fecundated ovule, and we may suppose that from an unfecundated one a monstrous polycystic growth should form.

Although out of the way, I wish to mention here that the only two cases in which I have been consulted on account of a return of abdominal cystic tumors, were where there was no pedicle, and where the chief vascular supply came from the vessels in front of the rectum. One case is the one now under consideration, and the other is the one published in the *American Journal of the Medical Sciences* for January, 1883, on account of the bladder having been opened during the operation.

My reason for reporting this case, however, is not to advance any particular views in regard to the pathology of certain polycystic tumors of the abdominal cavity, but in order to speak of the mistake made in the treatment. In the words of one who has done as much as any other to advance our knowledge of the treatment of abdominal tumors, "Mistakes teach most valuable lessons, and, when discovered, are not likely to be repeated. Hence, in medicine, they should be recorded for the benefit of science and of humanity." (*General and Differential Diagnosis of Ovarian Tumors*, by Washington L. Atlee, in Preface.) When this case presented itself, it was neces-

sary to act; a speedy and most painful death was inevitable unless relief were afforded. It remained to choose between the removal of the cyst and the emptying of its contents by tapping through the vagina, the only way practicable of introducing a trocar. On account of the operation previously performed, and the impossibility of moving the mass in the pelvis, its removal was not attempted. A previous operation is not necessarily an objection; Dr. Washington L. Atlee records two cases where a second operation was performed, and in both successfully. Spencer Wells, in his table, gives thirteen cases in which he removed an abdominal tumor from a patient who had previously undergone the operation, and eleven of them were successful. Here, however, the immovability of the tumor was such as to make me positively sure that the attachments were so great as to render its removal impossible.

Of tapping through the vagina I had no personal experience whatever, and what is recorded by others, although not, as a general rule, favorable to the plan, did not deter me from it. Scanzoni goes so far as to say that if puncture by the vagina was always possible, abdominal puncture would soon completely disappear from surgical practice. The sac, opened in its lowest part, can empty itself more completely. (Scanzoni, *Traité pratique des Maladies des Organes sexuels de la Femme*, Paris, 1858.) Peaslee says that there are three considerations to deter us from it in all ordinary circumstances: 1st. The vessels are larger and more numerous at the lowest part of the tumor; 2d. If polycystic, the largest cysts are not at its lowest part; and 3d. There is greater risk of wounding other

organs. (Peaslee, *Ovarian Tumors, their Pathology, Diagnosis, and Treatment, especially by Ovariotomy*, 1872.)

Emmet writes that he has punctured several cysts with a trocar from the vagina, and in every instance more or less cellulitis has resulted. This might, however, destroy the cysts, but he is not able to determine that, for all the cases passed from under his observation. (Emmet, *Principles and Practice of Gynæcology*, 1880.)

Thomas says that by this method two of the dangers of tapping, secondary escape of fluid into the peritoneum, and consequent peritonitis, are unquestionably avoided, but others are as surely increased, namely, those of injury to portions of the intestine and entrance of air into the sac, with consequent decomposition of the contents, septicæmia, and inflammation of the sac walls. His experience with the method is not large, but it leads him to agree with Spencer Wells. (Thomas, *Diseases of Women*, 1872.)

Spencer Wells says that tapping through the vagina is more liable to be followed by inflammation of the cyst than tapping through the abdominal wall, because it is not easy to prevent the entrance of air. The operation of tapping through the vagina is selected, not so much with the intention of simply emptying the cyst, as for the chance that should the fluid escape by the opening, as fast as it is secreted, the cyst may gradually contract, and the puncture close. This favorable result, however, is seldom secured. As a rule, air enters the cyst, the opening fills up, the fluid remaining in the cyst, or that newly secreted putrifies. Suppurative inflammation of the

lining membrane of the cyst comes on, and is accompanied by a low form of septic fever or pyæmia, which can only be relieved by making and maintaining a free outlet for the discharge. The frequency of these consequences should make tapping by the vagina an exceptionable practice, but it may be adopted in cases where an ovarian cyst is bound down in the pelvis by adhesions, and it is necessary to relieve the distress caused by pressure on the bladder and rectum.

The canula or an elastic catheter may be left in the cyst, though it is safer practice either to introduce a wire seton or a drainage tube, so as to prevent the opening from closing, and make sure of the free and immediate escape of any fluid that may be secreted. Even with every care, however, Mr. Wells concludes, from his experience, that patients are so apt to suffer from some of the ill effects of long-continued suppurative processes, that it is better, even at considerable risk, to remove a cyst, if at all possible, than to trust to any mode of drainage. (Spencer Wells, *On Ovarian and Uterine Tumors, their Diagnosis and Treatment*, 1882.)

Dr. Charles West, whose opinions have for me, at least, extraordinary importance, greater indeed on such a point than those of any other man, says that there is no doubt but that inflammation attacking parts within the pelvic cavity is less apt to extend to the peritoneum than when it attacks an abdominal organ; of which rule pelvic cellulitis and inflammation of parts within the folds of the broad ligaments are apt illustrations. It is equally incontestible that pelvic peritonitis is less apt to become generalized than is similar mischief originating in the abdomen.

Cyst inflammation is so dangerous, however, that it is doubtful if vaginal tapping may be employed, even when the cyst is simple. Its dangers are incalculable. The dangers of ovariotomy are far less. Except when a cyst is impacted in the pelvic cavity, he does not recommend tapping per vaginam. (West, *Lectures on Diseases of Women*, 1879.)

Our best authorities, therefore, teach us that the tapping of polycystic tumors at least must be avoided; that the removal of the cyst is to be practised in preference; that when impacted in the pelvis, so that it interferes with the performance of functions absolutely necessary to life, tapping, to which we must have recourse, is particularly dangerous, owing to the frequency of cystic inflammation, and consequent death from septicæmia. In my case I thought to avoid this by taking precautions to hinder the entrance of air into the cyst, but it took place notwithstanding, and the death of the patient was occasioned by it in eight days.

If, unhappily, I should ever again be called upon to try to relieve a similar case, I should act differently, and, not relying upon any means to prevent the setting up of cystic inflammation, do more to establish a free outlet for the noxious products by which the system is poisoned, and do everything in my power to prevent their formation by topical applications to the secreting surfaces of the cyst.

Dr. Noeggerath (*Ovariocentesis Vaginalis*, *American Journal of Obstetrics*, May, 1869), by pursuing this course, had such good results as to declare himself confident that, where a simple or compound cyst could be attacked through the posterior vaginal cul-

de-sac, this operation would take the place of laparotomy. The plan he prefers is to make first a transverse incision, about an inch long, behind the cervix uteri and through the roof of the vagina up to the cyst. This, coming into view, is then evacuated by a free incision, and, finally, the edges of the two incisions are stitched together by five or six silver sutures. The cyst thus left free and permanently open is daily washed out with antiseptic injections until it finally contracts, and ceases to afford any secretion. Although, at the present time, it is not likely that in ordinary cases any one would choose to perform this operation to that of the removal of the cyst, yet all experience shows that when the cyst is so firmly fixed in the pelvis as not to be removable, it diminishes in a marked degree the great danger necessarily incurred by tapping through the vagina—that of poisoning of the system by the absorption of the putrid products of cystic inflammation.

I feel certain myself that I made a sad mistake in not operating in this way, and report this case, as a warning to others—*Bene facit qui ex erroribus aliorum, sibi exemplum sumat.*

[After the reading of the preceding paper: —]

Dr. WILLIAM GOODELL said that he did not think the attachment of the tumor to the rectum was so unique as the return of the tumor. He had on several occasions removed tumors without being able to find the true pedicle, on account of the many adhesions. He could not agree with Dr. Atlee with regard to the origin of the cyst—he did not think it could have come from a wandering ovum, since, in that case, it would have been monocystic.

He regarded tapping per vaginam hazardous, for the reasons given by the late Dr. Peaslee, first, on account of the danger of wounding bloodvessels, and second, on account of the danger arising from the entrance of air into the cyst. He did not understand why the air could enter the cyst so readily after tapping per vaginam. When he performed tapping by this method he employed the aspirator. Returning to the question of the attachment apparently of the pedicle to the rectum, in the case reported, he said that it was quite common not to find a pedicle in some forms of intra-ligamentous cyst, because they had to be enucleated, and referred to the statements of Lawson Tait with regard to this difficulty of determining the origin of the tumor. He desired to call attention to the pathological characters of the recurrent growths, and stated that according to his observation they were always malignant, and that they recur in the stump of the pedicle, or, as he had seen, at the site of the adhesions. He alluded to a case which occurred in his own practice, and to one related to him by Dr. Bantock, of London, which confirmed the statements made.

He asked Dr. Atlee whether he could explain why the air entered so readily into cysts after tapping per vaginam.

Dr. ATLEE in reply said that he had seen no reason given, but thought it was owing to the difficulty in closing the wound made in the tapping.

Dr. R. P. HARRIS remarked that perhaps he could throw some light upon the cause of this admission of air, from what he had observed in a case of removal of hæmorrhoids. Some years ago, soon after the introduction of the écraseur of Chassaignac, he called in the late Dr. Joseph Pancoast, who had obtained the instrument, to make use of it in removing a mass of hæmorrhoids

from the rectum of a very anæmic man, rendered such by repeated hemorrhages after defecation. After the operation, to prevent, as he stated, the suction power of the diaphragm upon the rectum, endangering secondary hemorrhage, Dr. Pancoast introduced into the anus a small box-wood tube. The effect of this was soon demonstrated: a large drop of serous fluid descended the tube and stopped at its exit; when the diapraghm arose in expiration, the drop was drawn up the tube, and when it descended in inspiration, it came down again; thus showing the pumping effect of the diaphragm upon the pelvic contents. Let this open tube become an opened cyst with flaccid walls, and we have air pumped into the cyst, as it was into the rectum of our patient.

Dr. J. Ewing Mears asked Dr. Atlee whether a post-mortem examination had been made. He thought the observations of Dr. Goodell with regard to the difficulty of finding the true pedicle in tumors with many adhesions, and to the pathological characters of the recurring tumor in these cases to be correct, as such had been his experience in instances of which he had had personal knowledge. With regard to the entrance of air into the cyst, when tapping is performed per vaginam, he thought the explanations of Drs. Atlee and Harris were correct. A cyst firmly impacted in the pelvic cavity is separated from the vaginal tube by a comparatively thin septum, differing greatly in its anatomical characters from the abdominal wall. The opening made by the trocar in this septum and in the cyst wall attached to it by adhesions, does not close perfectly, and the air drawn up into the vaginal tube passes readily into the collapsed cyst.

Dr. Atlee, in reply to Dr. Mears, stated that no post-mortem examination had been made.

SEWER GAS, AND ITS ALLEGED CAUSATION OF TYPHOID FEVER.

By

GEORGE HAMILTON, M.D.

[Read March 7, 1883.]

The subject of the paper, now to be presented, is essentially similar to one read by the writer before the College in 1879, and reference to that paper will, from time to time, be made in regard to certain facts and statements therein contained. The opinion that sewer gas was, and continues to be, the principal cause of typhoid fever, and that all other causes, combined, are unequal to this single agent in the production of this disease, was opposed in that paper, as it will be in this. The views presented by Prof. Frank Hastings Hamilton, of New York, in a paper on "Sewer Gas," published in the *Popular Science Monthly* for November, 1882, are, essentially, those accepted by an immense majority, either in or out of the profession; and, on the other hand, they are either rejected, or not accepted, as a rule, by practitioners and writers who have had the most frequent and abundant opportunities for observing the origin, development, and progress of the disease. The important question then involved in this subject cannot be determined by mere numbers, but by an appeal to facts,

not conjectural, because theoretical, but to such as are unrefuted, because insusceptible of refutation. The great and increasing importance, in a sanitary point of view, of several questions connected with this subject, may be regarded sufficient to justify the expression of opinions in opposition to those entertained by one so deservedly eminent in the profession as the author of the paper on sewer gas.

In support of the sewer-gas theory Prof. Hamilton declares, "that the experience of every medical man, and of almost every intelligent citizen, is in accord." From an assertion in regard to medical men so sweeping as this, the writer cheerfully appeals to the page of medical history on this point; and the citizen must surely enjoy a rare degree of intelligence if he be capable of solving a problem in medicine that still remains *sub judice*. That the people in general have adopted the sewer-gas theory is beyond question; and the reasons for this are obvious. The efforts to gain over the public mind to this view have been, and continue to be incessant, and these efforts have, in great measure, been made by physicians of enthusiastic temperament, but who, unfortunately, have had but limited opportunities to obtain a personal, practical knowledge of this subject. But what is still more important, mankind are in general strongly disposed to accept as true that which promises pleasure, profit, and more especially health, exemption from disease and longevity. The latter have been freely promised, provided that the sewers, and the house connections therewith be properly constructed and kept in good condition; the filth in the streets, and every other place where it is apt to be found, be carefully removed,

and disinfectants properly applied. In this way the stamping out of typhoid fever, scarlet fever, and diphtheria was confidently predicted. Here, then, was the true balm of Gilead presented to view, and is it surprising that almost every one, even the incredulous, should banish the dread that had so long held them in doubt and apprehension, and turn with wistful and confiding looks towards, and gladly accept, the proffered boon; when, too, all this was to be had by the mere expenditure of the requisite money for a certain amount of mechanical and other labor.

A most discouraging thing, in this connection, is found in the assertion by Prof. Hamilton and others, that even when no sewer gas or offensive odor can be perceived, the gas with its fatal germs, may yet pervade the residence of an occupant, whilst he is ignorant of any method of finding out whether or not such germs infest his home. This asserted fact is, perhaps, the key-note to the proposition, that all the ordinary or extraordinary, and costly appliances now in use, and generally regarded, when kept in order, as capable of excluding sewer gas from dwellings, should be thrown aside, and that other apparatus should be erected in a detached structure, outside of the mansion. This proposition, emanating from a few sanitary engineers, is well-nigh impracticable. The plan has been tried in only a few cases, but not long enough to establish its supposed superiority. Colonel Geo. B. Waring is quoted in the paper of Prof. Hamilton as saying, "that such plumbing work as is to be found in nine out of every ten houses, even in Fifth Avenue, is unsafe;" what then must be the condition of ordinary houses consti-

tuting the vast majority, in view of the limited pecuniary resources of their occupants. Sanitary engineer C. F. Wingate is quoted to this effect, that the constant demand for the doctor's services in so many houses in their normal bad state; and the fact that his services are no longer demanded when they have been put in sanitary condition tells its own lesson. A lesson to have value, the Doctor forgets, must not be based upon assumed premises. From this quotation it does not appear that the apparatus was other than what is new in general use, but that it had "been put into sanitary condition," and in this he is in accord with a large majority of medical men and sanitarians. Dr. Barker, in his contribution to the paper on sewer gas, seems to deplore the fact "that of two young men, who suffered from typhoid fever, one died, and upon examination it was found that there was not a trap in the whole building!" But if the opinion of sanitarian Waring, and others who coincide with him, be correct, the mere existence of such apparatus within the walls of a building serves nine times out of ten, rather to increase than diminish the danger. The actual causation of disease, in general, is among the most obscure of medical problems; and in regard to the sickness of the Prince of Wales, cited by Prof. Hamilton, and also that in the National Hotel at Washington, or that occurring during the Centennial Fair, medical opinions are not at all in accord. Prof. Hamilton says "that plumbers suffer frequently;" but it has never yet been proven that they suffer more than others, although they are exposed to the action of sewer gas in its most concentrated form; and the reason why they do not suffer more, he informs us, is

"that they are in most cases in the full vigor of adult life and health." The reason here assigned will, the writer is satisfied, surprise every physician who has had an extensive experience in typhoid fever, or who is familiar with the written history of this disease. From early manhood, or womanhood, until the thirty-fifth year is the admitted period of by far the largest number of attacks, and this holds good in several other acute maladies, notably in bilious remittent fever. In regard to this disease the late Dr. Parish, eminent as a citizen and a most experienced and practical physician, said to his class of students, "that if any of them became country practitioners they would find bilious remittent very prevalent and fatal as compared with the city;" and, with emotion referred to his numerous consultations with country physicians within a radius of seven miles from Philadelphia, on either side of the Delaware, where he often witnessed the robust farmer, or his stalwart sons, sinking away into death, despite the efforts of the physicians. Now, that this form of disease is supplanted by the typhoid, a similar condition obtains, as may be seen when the writer states that four cases of intestinal perforation occurred to him in the space of twelve months, whilst practising in the country, thirty miles from Philadelphia. But more upon this point hereafter. Prof. Hamilton also speaks of the "immunity which adults usually enjoy, and especially those who are most of the time away from home and in the open air."

On the contrary, in his country practice, the writer nearly always found that the young men who were away from their residence, at work in the fields, were more frequently attacked with typhoid fever than the

females, who were generally engaged in domestic duties in or about the house; and if, as the sanitarians declare, "that there is in the country houses, or the surroundings, something equivalent to sewer gas," why is it that the reverse, as to sex, should not be the case? Diphtheria is also said to be caused in great measure by sewer gas; why is it, then, that the most violent and fatal attacks of this malady occur in the country, sweeping off at times four, five, or six children, in a single family, without a thought of sewer gas; whilst in cities, with sewer gas almost everywhere, the proportionate mortality is much less? Notwithstanding the testimony of Dr. Alfred Carpenter, of London, as quoted by the author of "Sewer Gas," to show the causation of scarlatina by this agent, here again such testimony is confronted by facts similar to those adduced in regard to diphtheria. It is only a few months since scarlet fever appeared in a town in Pennsylvania, noted for its cleanliness, and occasioned a proportionate degree of mortality unknown in Philadelphia. We now come to a singular and very important statement in the paper on "Sewer Gas," namely, "that in Asiatic cities, where modern improvements in plumbing are unknown, typhoid fever, diphtheria, and scarlet fever are seldom seen; whilst with us they have increased, as some declare, just in proportion to the extension of these improvements."

After this comes the admission of Dr. O. C. DeWolf, Commissioner of Health for Chicago, "that water-traps, as at present constructed, do not prevent the passage of disease-germs into our houses." Dr. Andrews is quoted by Prof. Hamilton essentially as

follows: "Canton is a city of bad smells, it has no water-closets or connection with sewers, and its *unsanitary* condition is unquestionable; yet there is rarely any typhoid fever or diphtheria;" and he expresses his belief, "that water-closets tend to propagate these diseases instead of preventing them." Prof. Hamilton declares "that the several classes of professional experts seem to have lost confidence in each other, and are heard constantly charging one another with incapacity." The chemists apparently are not agreed, the plumbers are charged with incompetency, and they, in turn, regard the physicians as their "most wrong-headed customers," possessing only a "dangerous smattering" of knowledge upon this subject. An architect has complained "that men of his calling have been blackguarded, lectured, and blamed by eminent doctors for their supposed ignorance of matters of this sort;" and a sanitary engineer has said publicly, "that there was probably only one architect in the city competent to execute the specifications for the plumbing of large houses;" while a distinguished member of the National Board of Health said publicly, "that he could count upon his five fingers all the sanitary engineers in this country in whom he could place any degree of confidence." If only the half of what has just been said be true, how embarrassing must be the condition of the public, and how greatly will be augmented the alarm, already so unnecessarily excited; and, in view of what has now been stated, may it not be thought that the motives thereto have not always been of a benevolent and purely unselfish character.

It is now time to confront the popular opinion that

typhoid fever, diphtheria, and scarlet fever are caused by sewer gas, and that to a degree unequalled by all other alleged causes combined. In attempting to do this, such cases as the following will be completely ignored. Mr. A., on returning from his summer tour, perceived, distinctly, the smell of sewer gas in his house, and fell sick on the third day with typhoid fever. What can be said against sewer gas in this case, it may be asked. In answer, it may be said, that while, by a bare possibility, the gas was the cause of the disease, the probabilities are all the other way. In the first place, the period of incubation is from eight to twelve days; in the second place, the writer's experience has convinced him that in not more than one house out of five has any smell or sign of sewer gas been detected, either before or during an attack of typhoid fever. And again, if the opinion lately advanced by a few of the most eminent authorities, that, as at present constructed and placed within the dwelling, the apparatus is incapable of excluding the gas, is correct, then, in such a city as Philadelphia or New York, many thousands more must be added to the tens of thousands of houses said long ago by the alarmists to be infected with sewer gas. In this connection, in may further be stated, that if the dwellings of this city and those of New York are infected to the extent asserted, then the conclusion is inevitable that the mortality would be absolutely frightful, and yet, in some weeks, in our city and in New York, with, taken together, about three millions of people, the deaths have been down to two, three, or four; while diphtheria caused, as sanitarians and inexperienced physicians say, by the same sewer

gas, has at this identical time occasioned weekly the death of about thirty or forty persons. In the third place, it is well known that many individuals on returning from the country to their homes in the city, in a very few days, before the period of incubation has expired, have had attacks of typhoid fever, and such cases may fairly be regarded as having had their origin in the country.

Another instance of typhoid fever is this: a complaint was made to the Board of Health, that a stagnant pond upon the outskirts of the city had already caused two persons to fall sick with fever. The pond was, by order, filled up, the patients recovered, and no others were attacked! Could anything, many would ask, be clearer than the exciting cause of these attacks? That a vast majority would regard the cause assigned as the true one need not be doubted; but it is not to be forgotten, that a large majority of people are by nature credulous, and, as a rule, the credulous are not logical, and the faith of such persons, as Goethe and Richter have said, is a matter of feeling, affection, aversion, rather than of reason, as the following example in relation to that just quoted, may serve to illustrate. Into the family of a wealthy farmer, a few miles distant from the ill-conditioned pond alluded to, typhoid fever entered at about the same time as in the former case; several of the members were attacked, and, as in the former instance, all recovered. There was no stagnant pond here to complain of; on the contrary, the farm lay in a beautiful elevated section of the country, was in all respects a model farm, the agriculture, domestic, and general economy exceptionally good; yet all this, as the

writer had often seen in his rural practice, occurred without the agency of sewer gas or (apparently) any other one of the agents so commonly held responsible for these attacks.

The writer is well aware that the points maintained in opposition to the current views as to the origin of typhoid fever are not accepted by a very large proportion of medical men and sanitarians; but it should be remembered, that by the reports of the Board of Health of Philadelphia, there have been on an average weekly, for the last ten or twelve years, but 7 to 8 deaths; hence, it is evident that, with all the alarm in regard to typhoid fever, there has not been opportunity for one-half of the regular physicians of this city to become fully acquainted with the disease, in its various forms and aspects. In every instance, however, when the writer has inquired of any one who had practised extensively, both in town and country, the answer has been nearly always, that the disease, when it appears in the country, is infinitely more prevalent and fatal than in the city, and that, as a rule, it was impossible to discover whence it originated. But, on this point, let us turn to an eminent authority—Trousseau, that great medical light of the nineteenth century. After speaking of the great difficulty of discovering the intimate causation of disease in general, and of the great advantages of the practitioner in rural districts in regard to this point, he declares, "that the ordinary causes given in explanation of the origin of typhoid fever are trivial (*banal*), and deserve no serious consideration."

Trousseau did not rashly, as often happens, declare his opinions on this point. His opportunities for the

study of typhoid fever in Paris were abundant, yet he was not satisfied with these, knowing, as he did, that the rural practitioner, having personal intercourse with nearly every family within a limited range, and consequently having superior advantages for observing the first outbreak and the progress of disease, would be most capable of arriving at a knowledge of its origin, whether from local or general agencies, or from contagion. To this source of information he often recurred, either by letter or personal interviews; and, endued, as he was, with an eloquence brilliant and comprehensive, a marvellous power of observation, and logical acumen, it is not strange that in life he was, and yet remains, in the first rank of philosophic medical authorities.

In illustration of the above statement my own experience may here be alluded to. Leaving Philadelphia in the autumn of 1834, a location was chosen at the distance of thirty miles from the city, between Wilmington and Kennet Square, seven miles distant from either, and about eleven from West Chester. The surrounding country was hilly, abounding in springs of fine water. Bilious remittent and intermittent fevers prevailed at this time, and for a few years after, in Philadelphia, to an extent that has not since occurred. The remittent form only held possession of this rural section, and before the frost of November put an end to the disease, as it always did. The statement of Dr. Joseph Parrish in regard to its prevalence and fatality in the country was fully confirmed in my own experience. After five or six years bilious remittent was supplanted by typhoid fever, and it would be inexcusable did the writer neglect to

cite two or three instances of this malady, of marked importance in relation to its origin and development. A wealthy farmer had sent a son on business into Maryland, forty miles from his residence. After an absence of several weeks he was brought home affected with typhoid fever, and, in succession, the father, two other sons, two daughters, and a colored servant were attacked, all severely, except the servant, the mother alone escaping the disease. An attendance lasting nearly four months was required, and, although three of the patients were in great danger, all recovered. In another family of seven persons, four were prostrated; one, a young man of eighteen years, dying from perforation. In this instance, again, the disease did not originate on the premises. The mother, the first attacked, had been absent from home assisting to nurse a relative, at a distance of several miles from her own residence, to which she was brought unwell, and thus communicated the disease to the others. Many physicians, perhaps all professed sanitarians, are in the habit of expatiating upon the great and unnecessary prevalence of what they called filth diseases: typhoid fever, scarlet fever, and diphtheria, in the large cities, and seem indisposed to turn their attention to the country until reminded, from time to time, of the ravages of these same diseases in rural sections, greatly exceeding, in proportionate number of cases, violence, and fatality what is to be found in cities. Yet the sanitarian, or the inexperienced practitioner, fertile in expedients, soon discovers the sources of their origin in decomposed vegetable matter in cellars and elsewhere, or in water and milk contaminated by the requisite family outhouse, while the attending

physician, though upon the spot, cannot, unless in exceptional cases, find any rational ground for these necessitated conjectures. But, in this connection, let us revert to the condition of agriculture and husbandry in regard to household, dairy, and stable economy half a century ago. At this period agricultural journals, or other works pertaining to those subjects, were scarcely known, whilst, for very many years past, they have been disseminated over the land, throwing a flood of light upon all that concerns agriculture and husbandry, the good effects of which are now visible on every side. It might here rationally be asked, What was the state of the country at the early period named, and was there not much more typhoid fever than has since occurred? On the contrary, typhoid fever and also diphtheria were almost unknown at that time, while bilious remittent, not generally regarded as a filth disease, prevailed, and so continued until about five years before the return of the writer to Philadelphia in 1845. As before intimated, a sharp frost or two would arrest the progress of bilious fever; not so in regard to typhoid; on the contrary, one of the worst invasions of the disease continued throughout the winter and early spring, and this peculiar and important feature finds its counterpart, not seldom, in this and other cities, where, independent of the agencies of warmth and moisture in promoting the decomposition of animal and vegetable matters, and evolution of gas, the disease has prevailed in an aggravated form.

In the writer's experience it did not often happen that either typhoid or remittent fever prevailed extensively in two consecutive years. In this respect it

resembled dysentery, raging with a fatality this year unseen in the city, whilst during the following season very few cases would appear—the local conditions in either case presenting no perceptible change.

An important point in the history of typhoid fever, as it occurred in the country, was, that if the disease once appeared in a farm-house, it would, as a rule, attack more than one of the members of the family before leaving it, and this, too, when the physician knew of no other case within a mile or more of the afflicted family. Now this is in entire accord with the admitted contagiousness, under certain conditions, of typhoid fever, not only by Trousseau, but by a majority of the most distinguished writers, among them that pre-eminent author and teacher, Austin Flint, Sr. The feature just noticed does not, as a rule, occur in the city, for in an immense majority of cases there will be but one member of a family attacked. Now it has been said by a somewhat noted medical statistician, and concurred in by many others, that if a case of typhoid fever appears in a family, it proves the existence of gas or filth in immediate relation with the residence. This is certainly explicit; but the question is, how happens it that, with such an efficient cause for the first case, it is only occasionally followed by others in the family? But, again, it may be asked, if sewer gas, for this, after all, appears to be the great "*cheval de bataille*," has not been, and is not yet excluded, by even the best plumbing, from the finest houses in this city or in New York, and, consequently, that tens of thousands of houses in both cities are in this fearfully exposed condition, how comes it that in Philadelphia, with 900,000 inhabitants, the Board of

Health has recently reported but two deaths from typhoid fever in one week, and four in another; whilst diphtheria, said to be due to the same agencies, has prevailed at the same time to an alarming extent? The returns of the Board of Health of New York present similar statistics, showing comparatively fewer deaths from typhoid fever than Philadelphia, but a larger proportionate fatality from diphtheria and scarlet fever. That many physicians neglect to carefully examine these reports is well known, and yet they are the only available and reliable sources of information in regard to the character and movement of disease. If, instead of this reprehensible neglect, a diligent and discriminating examination were regularly made, it would do much to prevent misconception and error in regard to the actual causation of disease, a subject admitted by the most experienced and gifted minds to be one of equal difficulty and importance.

In this connection it must be stated that the reports of the Board of Health of Philadelphia show, unmistakably, that in the central portions of the city, where sewers and water-closets are most numerous, there is, as a rule, less typhoid fever and diphtheria than upon the outskirts, adjacent to the country; and this was the fact in regard to bilious remittent fever, before it gave place to typhoid. Neither is it matter of surprise, for, just in proportion as the streets have been extended into the suburbs, graded and paved, houses erected thereon, and sewers constructed to carry off the surface drainage, a diminution of fever, whether intermittent, remittent, or typhoid, has been the beneficial result of such improvements. In support of this view we have the testimony of the late Doctor J. K.

Mitchell, the distinguished and brilliant Professor of the Theory and Practice of Medicine in "Jefferson Medical College," as follows: In December, of the year 1850, he was called to consult with the late Dr. Gebhard and the writer in a fatal case of typhoid fever, in a house on Sixteenth above Summer Street—at that remote period an outskirt of the city. On his first visit the doctor informed us that he was scarcely ever called upon to visit a patient affected with typhoid fever in a large circle of practice in his own section of the city, but that nearly all such cases were, like the present one, in the suburbs, and in consultation. Now the important fact in this connection is, that in his own central position sewers, water-closets, and the usual well in the yard, were almost everywhere, whilst in the suburbs they were comparatively few in number, the water-closet exceptionally rare. Does it not seem singular then, that, despite the improved, the scientific, the sanitary plumbing of the present day, "nine houses out of ten, on Fifth Avenue, New York, are not safe to live in;" and that all the actual appliances for the exclusion of sewer gas should be torn out, and others of an effective character be constructed in a separate building or annex? Dr. Mitchell also made allusion to the greater prevalence and fatality of fever in the country, as related to him by students from different sections of the United States.

In the paper by the writer upon this subject read before the College of Physicians of Philadelphia in 1879, Mr. Henry Dupont, in reply to a note, stated that his impression was that, in the years 1840–1–2–3, there were but few cases of typhoid fever among the

thousands of operatives and their families on the Brandywine, and this impression was corroborated by a note from Dr. Jos. P. Chandler, a practitioner of that section, in which he said that, whilst a very few cases *seemed* to owe their origin to local causes, his experience was perfectly in accord with that of the writer, that the disease nearly always showed itself in the houses of the farmers, and that no matter how well situated upon an eminence, with drainage in all directions from the residence, this did not secure them from attack. This is but a confirmation of the views long ago expressed by Dr. James Jackson, of Boston, and Dr. Nathan Smith, whose opportunities for observing the origin, development, and progress of the disease were exceptionally abundant. In a note sent to me a few days after the reading of the paper just alluded to, the writer declared that nothing was more erroneous than the current and popular opinions in regard to the causes of typhoid fever, and, in proof of this, cited the condition of the streets of one or two of the most populous and commercial cities of Africa, abounding, as they did, with every conceivable species of filth, emitting a stench nauseous to the last degree, and yet typhoid fever prevailed to a very slight extent. This statement is in accord with one quoted by Prof. Hamilton in his paper, before alluded to, to this effect, that in Canton, filthy beyond measure, typhoid fever and diphtheria are almost unknown.

In further support of these views, a physician of New England has informed the writer that a few years ago he addressed letters to about fifty practitioners, in different parts of the State in which he practised, asking their experience in reference to out-

breaks of typhoid. The answers, with very few exceptions, were, that the rural sections suffered the most, whether in regard to proportionate number of cases or fatality. Thus it now appears, as it did in the time of Nathan Smith and James Jackson, that typhoid fever outbreaks occurred without the possibility of referring them to the conjectural local agencies now in favor. Physicians who have not enjoyed sufficient opportunities to observe the origin and progress of typhoid fever, and sanitarians, without medical experience, are the most positive in opinion in regard to the primary causes of typhoid fever; whilst Trousseau, Flint, Sr., Murchison, and many others of similar experience and sagacity, freely admit the obscurity of this subject, and the difficulty of determining the influence of local agencies in the production of typhoid fever as compared with causes of a general character, over which we have no control, as in telluric or atmospheric conditions, involving thermometric, hygrometric, and electric states and their influence, singly or combined.

As pertinent to this point, reference may here be made to an article in the *Medical and Surgical Reporter* for January 1, 1881, entitled "The Limited Effects of Sanitation," from the pen of Dr. John Syer Bristowe, President of the Society of Medical Officers of Health, as follows: "If we look to the remarkable influence which simple variations of temperature and peculiarities of season exert on the mortuary returns, in respect both of the number of deaths and the character of the fatal diseases, and compare therewith the comparatively small effect on the death-rate of even one of the most fatal of the zymotic diseases, or with the

insignificant influence of deaths from enteric fever, diphtheria, pyæmia, puerperal fever, and other affections over which sanitary science is supposed to exert a specially valuable influence, we can scarcely avoid, I think, seeing that, on similar grounds, the deaths saved directly by the sanitary labors on which we are engaged must, under any circumstances, be so few annually as to produce no distinct and unmistakable effect on the mortuary rates." This sincere and ingenuous avowal of opinion by one so distinguished as an authority in medical science is well entitled to the serious consideration of the enthusiasts who never tire in predicting the suppression of the diseases in question so soon as sewer gas and filth are banished from the houses and premises of our citizens; whilst it is a notorious fact that lavish expenditures of money, by public authority and by citizens, have for several years past been made in Philadelphia without any perceptibly important result as the consequence. In the paper of Prof. Hamilton it is distinctly stated that, during a certain period, the increased mortality in New York could not be found in the condition of the streets, for at that time they were unusually clean; nor can the unusually severe outbreak of diphtheria now prevailing in our own city be attributed to the current local agencies. If, indeed, the primary causes of typhoid fever, diphtheria, and scarlet fever in cities be due to the influence of sewer gas and filth, then, in fact, our citizens can have little hope of an early exemption from these maladies. Yet, on this score, there is no occasion to despond. Fifty years ago typhoid fever was not common, but intermittent and bilious-remittent pre-

vailed in the city, yet only to a moderate degree compared to the suburbs and the adjacent country. These are not regarded as special filth diseases, whilst typhoid fever, diphtheria, and scarlet fever are by many so considered. At the remote period alluded to some attention was given to cleansing the streets, but there were no such exertions and expenditure of money, even in proportion to the size and population of the city, as are now customary; and as to inspection of dwellings, out-houses, wells, and the premises, it was almost unknown. That constant attention, by the authorities and citizens, should be given to maintain cleanliness in every place, and by all possible means, no one will deny; but, when all this has been done, what comparison can there be between the condition of a large city and any well-managed rural section in this point of view? Under the most favorable circumstances the sources of filth contamination in a city are as ten to one compared with the country, and this is manifest in the single fact, that the ground on which a city rests is, of necessity, well nigh riddled with wells for the convenience of families.

During the past ten or twelve years the average weekly deaths from typhoid fever in this city have been from seven to eight, as above stated. If this disease had prevailed here, in frequency of attacks and fatality, in proportion to population, as the writer has known it to do in the country, forty to fifty deaths per week would have probably occurred. The same feeling of gratification that the late Dr. Jos. Parrish manifested and expressed when informing his class of the slight mortality from bilious fever in this city, as compared with the country, has been experienced by

the writer in regard to typhoid fever in this city as compared with what is seen from time to time in the country. The city of Philadelphia, as shown by the reports of the Board of Health, still maintains its title as one of the healthiest of the large cities of the world, nor is it good policy, under any circumstances, to do or say aught to inspire citizens with dread, and induce strangers, who may have occasion to visit us on business, or otherwise, to regard Philadelphia as unhealthy, and therefore to be avoided.

[After the reading of the preceding paper:—]

Col. WARING, who was present by invitation, was called upon by the chair to discuss the subject considered in the paper. He remarked that he desired to say one word for sanitary engineers with regard to the cause of typhoid fever, and this was that they had long since abandoned the opinion that sewer-gas was the cause of the disease. He thought it very important that filth should be gotten out of the way so as not to become a source of contamination to the water-supply. The literature of the subject seems to prove that typhoid fever in the country is due to drinking-water which has become contaminated. Cleanliness is safe and important, and efforts should be always made to secure it.

Dr. HAMILTON said the quality of the drinking-water remained essentially the same from year to year, whether typhoid fever prevailed extensively, or was absent; so, also, in regard to vegetables and roots for family use. These are laid up every season, generally in trenches, covered with clean straw, and earthed over. On consulting several German monographs, Dr. H. had found the experience of the writers to conform with his own, that males suffered more from typhoid fever than females, and that from the fifteenth to the twenty-first year, the period of rapid growth, the danger, in case of an attack, was augmented.

Dr. J. M. KEATING said that the elaborate paper we have just heard read might mislead some of its hearers into the supposition that *sewer-gas* was considered a cause of typhoid fever. It may be well for us at once to dispel such an idea, and to couple with the paper just heard the emphatic endorsement of this body that *sewer-gas*, as far as *typhoid fever* is concerned, is simply a VEHICLE by which the specific germ may enter the system of one prepared to nourish and develop it. There is no need to debate the question as to the greater importance of infected drinking-water; the incidents in the paper just heard, the greater prevalence of the disease in the rural districts where privy wells contaminate the drinking-water, and the remarkable statistics of the New England country towns where the wells for sewerage and drinking purposes are under one roof, the fact that these localized epidemics are

usually traceable to one primary case entering the village, are all sufficient evidence in themselves.

But though *sewer-gas* may not always carry the germs of typhoid fever, it has a poison of its own equally terrible in its consequences.

The modern improvements in house-drainage—indescribable comforts where properly constructed—are unfortunately too often the means of carrying the most deadly vapors directly into the abodes of luxury and wealth. At these hours, when depressing influences have disarmed their victim, these noxious gases steal insidiously into his chamber and do such violence as to render fatal by the most serious complications such diseases as are otherwise mild and harmless in themselves. Whether the concentrated miasm from polludial emanations or the outpouring gases from a sewer through siphoned traps, the unconscious sleeper breathes poisons as potent in their devitalizing effects as if he inhaled the germs of typhoid fever in their purity or drank water impregnated with the excreta of typhoid cases. *Sewer-gas* is not necessarily a *cause* of typhoid fever, except when it carries its germ, but it is the most potent cause of the "typhoid state;" and there is a decided choice between these two evils—in favor of the former.

Early manhood carries with it a decided predisposition to typhoid fever. I have recently read with great interest an article in the *Nineteenth Century*, by Gen. Sir Frederick Roberts, on the recruits for foreign service in the English army, in which statistics were given to show that the large mortality from typhoid fever, in young recruits, rendered advisable the considerations as to whether it was not better for foreign service to take only such as had recruited early and survived this period, or to recruit only from those who had passed the age when the disease was most liable.

The typhoid germ is certainly more deadly if its vehicle be poisonous also; and it becomes our duty to add an earnest appeal to that which seeks by proper legislation to give us pure air for our children to breathe and pure, uncontaminated water to drink.

Dr. J. G. Richardson remarked: I am well aware that the evening is too far advanced for me to attempt to discuss the important subject of Dr. Hamilton's paper, and I will therefore

content myself with suggesting how two of Dr. H.'s illustrative cases may be very plausibly explained in accordance with the modern views respecting the danger to human health from impure drinking water, foul sewer-air, and other grave sanitary errors.

Dr. Hamilton mentioned the case of the mother of a family who contracted typhoid fever away from home, and brought it back to her farm-house, where several members of the family seemed to take the disease from her, and argued that this circumstance proved that the infection of typhoid fever was not carried by sewer-gas, because in this instance no sewers existed near the place. The true explanation of this seeming anomaly is, I believe, however that, whilst typhoid fever is sometimes carried by sewer-gas, it is more frequently conveyed in drinking water contaminated by sewage, and that in this case the diarrhœal discharges of the mother were emptied near enough to the family well to soak through the ground into it, and, by polluting the drinking fluid, transmit the disease to other members of the household.

That some such mode of propagation may have existed is shown by a remarkable fact related by Prof. Flint upon whom Dr. Hamilton relies as an authority against the dangers of sewer-gas, which is briefly as follows: A young man travelling by stage coach in Vermont was taken sick, and left at a wayside inn in a small village. His complaint soon proved to be typhoid fever, and in a short time the disease appeared in each of the neighboring houses, the inhabitants of which used water from the tavern-keeper's well, *except one*, whose residents had quarrelled with the landlord, and consequently went elsewhere for their water supply.

In the light of recent sanitary science, then, it appears that many people, many whole families, in rural districts, suffer from typhoid fever because they drink well water polluted by direct mixture with fecal matter containing the specific fever germ, which soaks through the soil, often directly, into the well; whilst in cities a few persons, relatively, are attacked with typhoid fever, and many with diphtheria, etc., by inhaling poisoned sewer-air from stationary wash-stands and other modern conveniences. There are probably one hundred thousand village and farm-houses in the United States to-day where the cess-pools *feed* the wells in this

abominable manner, and such wells surely in their turn *feed* from time to time the churchyards in their neighborhood.

Again, Dr. Hamilton inquires, why is it, if sewer-gas is the cause of both typhoid fever and diphtheria, we now have, according to the late reports of the Board of Health, only two or three deaths per week from the former disease, and thirty or forty from the latter? The obvious answer is that sewer-gas can only convey the *materies morbi* (the disease germ as I believe it is) with which it is freighted, and at the present time sewer-air in but few houses is contaminated with typhoid-fever poison, whilst in many it is loaded with diphtheria germs. Precisely as drinking water might contain both lead and copper in varying proportions, and accordingly as one or the other temporarily predominated, would persons who imbibed the fluid be liable to exhibit the symptoms of lead palsy or of copper poisoning.

Although I am not yet prepared to endorse the *dictum* of a late English hygienist, who declares "For every death from typhoid fever, somebody" (meaning some plumber, architect, or sanitary engineer) "ought to be *hung*," I do hope and believe that the time is not far distant when at least the charge of criminal negligence will lie at the door of every physician who, after being warned by the occurrence of one case of typhoid fever, diphtheria, etc., in a dwelling, does not immediately endeavor to guard the other residents of that house against the germs (or poison, if you choose) of infectious disease, by a diligent search for the sanitary defects in the water or milk supply, or in the disposal of sewage, which will almost certainly be discovered.

I have spoken of the germs of typhoid, diphtheria, etc., entering sleeping apartments from stationary wash-stands in spite of traps, and in this connection I desire to show the Fellows of the College just how, as I conceive, these poisons may penetrate through such mechanical contrivances. Here I exhibit the ball of an ordinary Bower trap, covered with a coat of fungous growth, and here a portion of the slimy coating from one of the pipes of similar structure. Since these form a continuous lining quite through the trap, it is obvious, I think, that it may have crept up from the sewer itself, thus forming on the inner or house side of the trap a new starting point for the evolution of disease germs, against which of course the most perfect water seal is absolutely no protection. This, I believe, is what really occurs

in a multitude of instances, as I pointed out in a paper entitled "Why Sewer Traps are Unreliable" in the *Medical News* for September 2, 1882. The true remedy for the dangers of sewer gas poisoning is, therefore, I think, as explained in that article, to be found in sterilizing the whole interior of our traps and waste pipes with slow currents or drippings of powerful disinfectants, such as the salts of iron, zinc, mercury, or arsenic, "just as the shores of the Dead Sea and the banks of certain small streams are sterilized, by mineral ingredients or poisonous metallic substances from manufacturing refuse, with which their waters are mingled."

TUBERCULAR CEREBRO-SPINAL MENINGITIS.

By

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[Read April 4, 1883.]

In the year 1768, Dr. Robert Whyt, of Edinburgh, in a small brochure, described the most common form of acute hydrocephalus, directing attention to the connection between acute inflammation of the meninges of the brain and dropsy of the ventricles. (*Observations on the Dropsy in the Brain*, 8vo. Edin. 1768.) About half a century later, French anatomists showed that in the majority of the cases of Whyt's disease, the membranes of the brain themselves were the seat of tubercular deposit. (*West on the Diseases of Infancy and Childhood.*) In the year 1825, Marshall Hall described the hydrocephaloid disease. Soon to his accurate observations were added those of Gooch and Abercrombie. In this disease, which is also called by Watson spurious hydrocephalus, no inflammation is supposed to exist, although more or less effusion is found in the ventricles. Fine granulations on the cerebral meninges, often unattended by inflammation appreciable to the unaided eye, had been observed for a long time, but their exact nature

was unknown until the year 1830, when Papavoine showed them to be tubercles. (*Journal Hebdomadaire* for 1830, vol. vi. p. 113, quoted from West.)

The literature on the subject of tubercular cerebro-spinal meningitis is exceedingly meagre, many writers on tubercular meningitis not alluding to it. Dr. Samuel Jones Gee (Reynolds's *System of Medicine*, edited by Hartshorne) states that he has several times examined the cerebro-spinal opening *in situ*, and has always found the membranes about it perfectly healthy. The spinal sub-arachnoidean space was distended with fluid, especially around the cauda equina. He never observed any other morbid condition within the spinal canal in persons dead of tubercular meningitis. It must be remembered that he examined the cord in a minority of cases.

About the year 1869, three observers, MM. Magnan, Hayem, and Lionville, published almost simultaneously cases of tubercular cerebro-spinal meningitis, giving as their opinion that tubercles occurred at the same time in the membranes of the brain and cord. The special signs which they attributed to this disease were, tremblings, contractures, tossing, restlessness, tetanic seizures radiating to the neck and trunk, and temporary paralysis. Their autopsies revealed lesions of the cerebral membranes, and granulations on the surface of the spinal pia mater and on the arachnoid. Once the dura mater was most affected and fibrinous exudation was present. (*Le Progrès Médical* for 1881, Galliaux.)

Flint (*Practice of Medicine*, fifth edition, p. 702) says: "Tuberculosis of the pia mater of the spinal cord has been found in many instances, and, probably,

is the rule." He, however, does not appear to have met with a case in connection with tubercular meningitis.

Huguenin in his elaborate article on tubercular meningitis (Ziemssen's *Cyclopædia of the Practice of Medicine*, vol. xii. p. 505) disposes of this disease in the cord in one short paragraph, as follows: "Our knowledge here is quite fragmentary. It is certain that tubercles are found in the spinal cord in many cases of tuberculosis of the pia, and also that their behavior is the same as in that of the brain. The inflammatory affection of the pia seems to pass down a varying distance within the canal. There are no trustworthy statements as to the changes of tissue in the spinal cord; but without doubt many symptoms would, after a more careful investigation of this subject, appear in an entirely different light from that in which they do now."

In the *Transactions of the Pathological Society of London*, in vol. ii., Mr. Shaw reports a case of tubercles of the brain, and of the spinal marrow and its membranes. The patient was paraplegic, but conscious to the last. In vol. xxi. of the same Transactions, Dr. Walter Moxon reports miliary tubercle of the spinal dura mater occurring in a case of tubercular meningitis. The patient was a girl, æt. 17 years. Duration of the disease was seventeen days.

In the *St. George's Hospital Reports* for 1879, fifty cases of general tuberculosis are analyzed. In a large number of these brain lesions were found, but in only one instance was the spinal cord or its membranes found diseased. It is not stated in how many instances the cord was examined, but that spinal lesions

were unsuccessfully sought for in cases of tubercular meningitis appears from the report. The case in which the spinal meninges were involved, occurred in a seven and a half months' child, male, æt. 4 years, of strumous diathesis and of a consumptive family. The mind was precocious, and the skin dry. The disease began with cough; headache after about two weeks, when the cough almost ceased. The duration of the lung trouble was sixty-one days; of the head about forty-seven. Tubercles were found in lungs, pleura, and spleen (?); on the meninges of the brain and arachnoid of cord, and a nodule was seen on the under surface of the cerebellum.

H. Rendu (*Recherches Cliniques et Anatomiques sur les Paralysies liées à la Méningite Tuberculeuse*, Thèse de Paris, 1873), Landouzy (*Contribution à l'étude des Convulsions et Paralysies liées aux Méningo-encéphalitis fronto-pariétales*, Thèse de Paris, 1876), and Chateaufort (*Contribution à l'étude de la Méningite Spinale Tuberculeuse*, Thèse inaugurale de Paris, 1878, No. 384), in the years 1873, 1876, and 1878 respectively, added contributions to our knowledge of the subject of tubercular cerebro-spinal meningitis. (*L'Union Médicale* for 1879, Debove.)

Debove (*Le Progrès Médical* for 1879) reported a case of tubercular cerebro-spinal meningitis. The patient was a man, æt. 29, suffering from pulmonary phthisis. He suffered from severe lumbar pains and unsteadiness of gait for about two and a half months; paralysis of left leg and inability to void his urine three days; paralysis of both legs, insensibility of the left and partial insensibility of the right two days.

Delirium was present for the first time about twelve hours before death.

At the autopsy tubercular granulations were found on all the membranes of the cord, most abundant on the pia mater near the anterior and posterior fissures of the cord. Congestion was intense in the lumbar region, and suppurative meningitis was most marked posteriorly in the dorsal. Tubercles were sparse and congestion slight in the cervical region. In the brain a few tubercular granulations were seen along the fissures of Sylvius, with little congestion of the meninges, without a trace of suppuration or fibrinous exudation. The cerebral substance was normal, and the ventricles did not contain an abnormal quantity of fluid.

Dr. Debove calls attention to the following facts in connection with his interesting case: First, the primary lesion was in the spine, the brain becoming secondarily affected, the reverse of what usually takes place. Second, the principal phenomena during life were due to the spinal rather than the cerebral lesion. In the first report of this case, he called it tubercular cerebro-spinal meningitis; in his second, before the same society, a few months later, tubercular spinal meningitis.

Galliaux (*Le Progrès Médical* for 1881), after referring to the observations and conclusions of MM. Magnan, Hayem, and Lionville, gives a short account of a case of tubercular cerebro-spinal meningitis coming under his care. The patient was a man, and like that of Debove's was 29 years old. He was brought to the hospital in a semi-conscious condition two days before his death. His wife stated that he

had suffered from cough six weeks, and a few days before being brought to the hospital the cough ceased, and at the same time he began to suffer from fever, diarrhœa, and epistaxis, symptoms which his medical attendant had attributed to typhoid fever. Galliaux detected well advanced tuberculosis of the lungs. The autopsy revealed, in thorax and abdomen, tubercular infiltration of the lungs with small cavities at the apices, pleuritic adhesions, extensive ulcerations in the intestines; in the brain, a normal dura mater, a pia mater presenting adhesions to the brain substance, most marked around the tuber cinereum, and on the internal surface fine tubercular granulations, most abundant in the fissures of Sylvius. No pus nor free fibrinous exudation was found. The meninges were not thickened or much congested, presenting nearly their normal transparency. Some serous fluid was around the tuber, and a similar fluid filled the lateral ventricles. The cerebral substance was soft, but presented no appreciable lesions; in the spinal canal, the parietal and visceral layers of the arachnoid membrane were adherent, the bloodvessels of the dura mater were injected, evidences of slight inflammation were present, and fine tubercular granulations were found on its internal surface.

L. Dubar's (*Méningite Cérébro-spinale Tuberculeuse*, *Bull. Soc. Anat. de Par.*, 1879, 4 s. iv. 240–243) contribution to the subject I was not able to obtain, nor were the results of the investigations of Von-Azary accessible. (Von-Azary (A.) *Beiträge zur Turberkulose des centralen Nerven-systems der schweine Deutsche Ztschr. f. Thiermed.*, Leipz. 1880, vi. 254–269.)

As the following case presents many features in

striking contrast to the phenomena exhibited by those already reported, I venture to record it, with as concise history as possible, containing numerous general and surface temperature observations, in the *Transactions* of the College.

H. J., æt. sixteen months, was moderately well nourished but excitable. A superficial suppurating gland had existed on the anterior portion of the neck since the early months of infancy. The father, about thirty-five years old, a German, has a strong and sturdy constitution, and gives an exceptionally good family history. The mother, also of German parentage, is thin, anæmic, and painfully nervous. So far as she knows, her ancestors were free from phthisis and scrofula; her father and mother, about seventy years of age each, are living and well. Some of her brothers died from pulmonary consumption, apparently induced by exposure and dissipated habits.

When about ten months old, the child had an irritative fever, apparently from teething, lasting four or five days. During the morning of April 15, 1882, about six months after suffering from the fever, it was apparently well, and spent a portion of the time in playing about the yard, but late in the afternoon it became feverish, fretful, and refused to eat. The symptoms grew worse, and I was called at noon the next day. I found undoubted evidences of alarming illness attended by great prostration. Ten or fifteen dark blotches of extravasated blood were seen on various portions of the body; the head was somewhat retracted, and the eyes were turning from side to side; temp. 103°; pulse 150; resp. 84. The central incisor teeth only were erupted, but the gums covering the lateral incisors were swollen and painful. These were freely lanced, and the child placed in a hot bath and given potassium bromide. Grave brain trouble was suspected. At 4 P. M., pulse 180; resp. 96. A violent tetanic convulsion with occasional clonic movements took place during the examination. The face was extremely

pale and the child thought to be dying. The convulsive seizure lasted in its worst form about one hour, during which time the little patient lay immersed in hot water. After the fit passed off, the left arm and leg remained rigid, the hand being shut and the foot extended until 4 A. M. the next day. The first evening of my attendance the temperature fell to 102.6°; the pulse varied from 160 to 180, and respiration from 90 to 96. The hemorrhagic extravasations under the skin disappeared when the body was immersed in warm water. Beginning on the morning of the 17th with temperature of 101.5°, pulse of 160, and respiration of 84 per minute, the symptoms gradually ameliorated during that and most of the two succeeding days.

April 19. Morning, temp. 100°; pulse 118; resp. 50. It was still restless, refused to eat, cried and moaned most of the time, and slept in short naps only. 8 P. M., temp. 103.8°; pulse 135; resp. 84. The change was sudden, the child apparently being much worse. A cough beginning about that time was the only evidence of chest trouble. Twenty-four hours later the resonance of the apex of the left lung was noticed to be much impaired, but no rales could be detected. On the 20th the child slept a good portion of the time. From the 20th to the 24th, it took but little nourishment, vomited large quantities of phlegm, and incessant cough attended by large mucous bronchial rales, heard all over the chest, kept the little patient fretting. The temperature ranged from 101.8° to 103.5°; pulse from 112 to 140; respiration from 40 to 60.

24*th*. 11 A. M., temp. 98°; pulse 120; resp. 48. 6 P. M., temp. 105°; pulse 130; resp. 60. From the 24th to the 29th, the daily range of temperature was from 98° to 100°–103°, the rise being as often during the morning as the afternoon hours. The pulse varied from 130 to 140, and respiration from 68 to 70. On the 29th, when Cheyne-Stokes respiration was present, the temperature did not descend below 101°, and reached 102° at 6 P. M. In the evening, Dr. Charles K. Mills saw the case with me, and we agreed that

it was probably tubercular meningitis of an irregular type, although the peculiarity of some of the symptoms made us doubtful as to the accuracy of the diagnosis.

On the 30th the morning temperature was 98°; pulse 116; resp. 50; evening, temp. 104°; pulse 170; resp. 70. The pupils were still small, the head was again retracted, and the child kept up a pitiful moan. During the afternoon, Dr. T. G. Morton met me in consultation. He thought it was a case of tubercular meningitis. Every effort was made to keep the child nourished. In the way of medication it was given the potassium iodide and bromide, and small doses of calomel, or corrosive sublimate. When the bromide failed to relieve, twenty drops of the camphorated tincture of opium were administered every hour until quiet was produced. Cold was occasionally applied to the head, and mustard, from time to time, to the nape of the neck.

May. During the month the highest axillary temperature was found on the 11th, in the morning, when the thermometer registered 105.5°; the lowest was taken on the 1st, also in the morning, and was 95.6°. The exacerbations of fever were very irregular. On a few occasions during the month, the temperature rose to 103° to 105°, and descended to normal or below the same day, but the febrile paroxysms, however, extended over a period of twenty-four to thirty-six hours, and the lull, during which the temperature was normal or subnormal, lasted from twelve to twenty-four hours. Twice the period of heightened temperature with remissions lasted six days (from the 6th to the 11th, and the 26th to the 31st, inclusive). Once only (on the 15th, 16th, and 17th) did the temperature remain normal or below a period of three days. Throughout the month, the fever had marked remissions which were always attended by free perspiration, simulating, in this respect, malarial remittent fever. The pulse and respiration were frequent, being most rapid, as a rule, when the temperature was highest. The pulse range was from 116 to 180 per minute, being 170 on one occasion, when the temperature was only 96.5°, and fre-

quently having a rate of 150 to 160 with a normal or subnormal temperature. The frequency of the respiration varied from 36 to 86 per minute, the average being about 60. Those interested in a complete record of the temperature, pulse, and respiration, are referred to the tabular view of this case.

The posterior muscles of the neck were quite firmly contracted on the 5th, and remained so nearly two days. From the 1st to the 8th, the child was very restless, and required repeated doses of camphorated tincture of opium. On the 9th, it became more quiet, but semi-choreic movements of the muscles of the neck, face, and upper extremities, when it was awake, were noticed. Those movements at that time lasted parts of two days only. During the entire day, on the 11th, the child was drowsy, and could scarcely be awakened, although it had taken nothing to induce sleep the previous two days. It would drink, however, when milk was poured into its mouth. From April 16 to May 11, the pupils had been rather small, and often very much contracted, but subsequently to the latter date they were noticed to dilate, sometimes to their full extent, just before and during a paroxysm of head pain. On the 14th, cough, which had been absent nearly two weeks, returned, and was more annoying than during the first attack of pulmonary trouble. Numerous subcrepitant rales, most abundant in the upper portion, were heard in the left lung. On that day, seven and a half grains of quinia were given in divided doses. In this daily quantity it was continued for a period of two months, with the exception of two or three days, when it was temporarily suspended. From the 13th to the 31st, the child was very restless, keeping up an almost constant cry, and apparently suffering great pain. Opium was the only thing found to give relief. On the 18th, the choreic-like movements returned and continued a day or two. On the 19th, about the time that the lateral incisor teeth were erupted, the left ear began to discharge considerable non-offensive pus. The next day eight twenty-drop doses of

camphorated tincture of opium were given at hour intervals before rest was obtained. On the 22d, when the muscles of the back and right side of the neck were contracted, ten thirty-drop doses of the same were given at equally short intervals; and on the 23d, fourteen and a half drachms, or nearly two ounces, of this preparation of opium were administered without entirely quieting the child. The discharge from the left ear, still yellow, was thinner and more offensive. It soon became exceedingly unpleasant; by the last of the month the ear ceased to discharge. Instead of the camphorated tincture of opium, morphia was subsequently employed, and gradually increased; the quantity within two weeks necessary to quiet the child, during some of the nights, being two and a half grains. During the afternoon of the 31st the little fellow was bright, free from pain, and quite playful.

June and July. The temperature during these months ran a less variable course, reaching 104.1° only once (June 9), and never descending more than a degree and a half below the normal. The average temperature for the two months was about 99°. The pulse range was greater, the frequency being 180 on a few occasions, and once (July 8) as low as 86 per minute. When the pulse was slow it became intermittent.

June 5. The head was again retracted, large doses of morphia being necessary to afford relief. About that time the child became very passionate, screaming, and striking at every one (except its mother) who came near it. It was conscious and rational, and would promptly answer in the affirmative when asked if it wished to be taken out in its coach.

8th. The left ear was again discharging non-offensive, thick, yellow pus. A diffuse bronchitis with numerous mucous rales, causing great oppression in breathing, set in about that time and lasted three or four days.

On the evening of the 11th of June, with widely dilated pupils, the child began to scream, and continued to cry vigorously, manifesting other expressions of pain four or five hours, notwithstanding four doses of one-third of a grain of morphia each were administered at short intervals. The

next morning it seemed to be free from pain, but was not sleeping continuously.

14*th.* Both ears were discharging quantities of yellow non-offensive pus. No teeth were about to be erupted, the gums not being swollen. From the first to the fourteenth of June, the little sufferer, when not under the influence of morphia, was almost constantly screaming.

About the middle of June, it became quiet and ceased to cry except when disturbed. During the latter half of June, and the entire month of July, no anodyne was required. When the administration of large quantities of morphia were necessary the axillary temperature was only exceptionally above 99°.

19*th.* A diarrhœa began and lasted a few days, the food passing through the bowels undigested.

July 1. I began to register the surface temperature of the head, not having ventured before because I feared the irritability and restlessness of the child would endanger the safety of the thermometers (two thermometers always being used at the same time).

On the 8th, the pulse was slow and intermittent, the stomach irritable, and the bowels loose. It was noted that the child was decidedly worse every second day, being feverish some time during the twenty-four hours on alternate days. The fever was always followed by free perspiration. During the month the little patient seemed to improve, and it was taken into the open air every clear day, and sometimes into Fairmount Park during the early morning hours.

August. From the 1st to the 15th, the child was quiet, and did not fret when left undisturbed, the temperature ranging from 97.5° to 99.5°.

On the afternoon of the 16th, it suddenly became convulsed, was rigid, and remained in this condition about ten minutes.

18*th.* There was some twitching of the muscles of the extremities, especially of the hands and feet. The breathing was the Cheyne-Stokes variety. One forty-eighth of a grain

of morphia was required to relieve pain, it being the first day that an anodyne had been necessary since June 1st, a little more than two months. Increasing quantities of morphia were required almost daily the remainder of the month, the child being feverish and restless every afternoon, and frequently screaming violently from head-pain. The range of temperature for the last eight or ten days of the month was from 100° to 102.5°.

On the 25th, 26th, and 27th, Cheyne-Stokes respiration, minus the intermission, was present; and on the 28th, typical Cheyne-Stokes breathing lasting one day only.

September. My notes of the case for the 1st, 2d, and as late as 4 P. M. of the 3d of the month, show a normal axillary temperature, and fairly good pulse and respiration, but the surface temperatures of the head at the 4 P. M. examination of the 3d were 1° higher than the axillary. After having been comparatively quiet for nearly two months, and resting well during the previous two days, it began to cry with apparent head-pain. The pupils were widely dilated. It screamed furiously, and almost immediately, while the thermometers were still on the head, became convulsed. At first every muscle appeared to be rigid, the posterior muscles of the neck and those of the back being most contracted, and producing an extreme condition of opisthotonos. The eyes rolled from side to side, and a few spasmodic movements of the muscles of the body took place. The spasmodic seizure lasted about half an hour, but the leg muscles remained rigid much longer. Respirations were 68 and the pulse 130. At 9 A. M. the next day it had another convulsion, which soon passed off, leaving a number of muscles contracted. At 10 A. M., I found the muscles of the back of the neck, and the flexors of the legs and arms, and the extensors of the feet, firmly contracted. Twitching of the facial muscles took place occasionally, each spasmodic contraction of these muscles being attended by a scream from the child indicative of great suffering. Temp. 103.5°; pulse 148; resp. 16. The head temperatures were a half degree below the axillary. The

axillary and head temperatures continued to rise until the former reached 104° and the latter 103.5°. The condition of the muscles remained nearly the same during the day, except those connected with the eyeballs, which became more affected, and kept up a continuous nystagmus. The respirations became very slow and irregular, the pause often being from ten to fifteen seconds in length. 10 P. M., temp. 103.5°; pulse 140; resp. 8. Numerous bronchial rales were heard throughout the chest. During the next two days the temperature varied from 102.5° to 103°; pulse from 140 to 150, and respiration from 28 to 34. The spasmodic movements of the facial muscles ceased, the back muscles of the neck and the flexors of the arms relaxed, but the adductors of the legs and the extensors of the feet remained contracted, and so continued gradually increasing until the death of the child, nearly three months later. The following week the temperature ranged from 97 to 100.5°, the average being about 99°. The axillary temperature was frequently lower than the surface temperatures of the head. The breathing was of the Cheyne-Stokes variety once or twice, and on a few occasions it was slow and irregular. The head was retracted about one-half the time, that condition always being associated with symptoms of pain, irritability, and heightened general and surface temperatures. Small doses of morphia (one-fourth to one-eighth of a grain) were sufficient to quiet the child.

13*th*. 10 A. M., temp. 93.8°; pulse 96; resp. 14. The head temperatures were about 5° higher than the axillary. After that date, to maintain the body heat, it was necessary to have bottles filled with hot water, and to keep them constantly applied to the extremities. The remainder of the month the temperature did not descend below 95°. The deformity of the legs and feet became more marked. The legs were forcibly crossed near the body, and the feet were extremely inverted and extended. A straight line drawn from the anterior surface of the knee to the upper surface of the tarso-phalangeal joint of the great toe passed through the instep, and one drawn from the popliteal space to the under

surface of the same joint of the toe just touched the under surface of the heel. The child was peevish and fretful most of the time. An afternoon rise of temperature was the rule.

On the 28th, the biceps muscle of the right arm was noticed to be contracted. The next day it was more flexed, and the pulse was intermittent. The head became so much retracted about the latter part of the month that deglutition was very difficult. To relieve that condition an issue was established by means of Vienna paste over the upper portion of the cervical spine. After this the head was rarely retracted, and then only to a slight degree.

October. The general condition of the child continued about the same, the right arm still being flexed. On the 6th, the left arm was first noticed to be affected, the flexor muscles of the forearm being contracted, and the hand deflected to the ulnar side. From that date the arm, like the leg muscles, continued to become more firmly contracted. The right arm was drawn in front of the chest and firmly held against it, the forearm being flexed so that the closed fist was under the chin, making it necessary to cover the hand with cotton to prevent its interfering with respiration and deglutition. The biceps muscle of the left arm was never firmly contracted, the flexors of the left forearm being mainly affected. The left hand was forcibly flexed upon the forearm, and turned to the ulnar side, the finger-nails striking the first phalanx of the thumb. The palm of each hand was padded with cotton to prevent injury by the nails. During the month the axillary temperature varied from 98° to 101.2°, the average being nearly 100°. The head temperatures were a little lower than the axillary. On the 10th, both eyes were turned to the left, and occasionally moved spasmodically downward and far over toward the left side. The eyes remained deflected toward this side, but at times they were nearly straight. Spasmodic movements of the eyes in various directions were noticed when the child suffered much pain. More or less morphia, sometimes a grain

in the twenty-four hours, was required the greater portion of the time. From the 18th to the 29th, respiration was of the Cheyne-Stokes variety, the child being restless and suffering pain most of the time. Occasional spasmodic twitchings of various muscles of the body were noticed, those of the face and arms being most affected. The clonic, spasmodic movements seemed to be caused by increased cerebral irritation, and were almost invariably followed by screams from the little sufferer. At those times the permanent contractures were worse. On the 24th, two grains of morphia were given before pain was relieved. The child had been noticed to stare meaninglessly for several days, but it was not found to be entirely blind until the 29th. At that time an ophthalmoscopic examination of the fundus of each eye showed both optic disks to be very white, and apparently slightly swollen.

November. The first twenty days of the month the child was comparatively quiet, ate well, seemed to gain some flesh, and was generally quiet without morphia. During that period it was most irritable the 3d and 11th, but it did not have much rise of temperature on those days. The child was worse every second day.

21*st* and 22*d*. There were numerous bronchial rales, but no fever, the breathing seeming to be a little more labored than usual.

23*d*. Temp. 98°; pulse 108; resp. 28. Large bronchial and subcrepitant rales abundant.

24*th*. 10 A. M., temp. 96.7°; pulse 130; resp. 40. 6 P. M., temp. 99.6°; pulse 140; resp. 52. Impaired resonance posteriorly; crepitant and subcrepitant rales were observed.

25*th*. 10 A. M., temp. 101°; pulse 160; resp. 80. Marked dulness was found over the lower portion of both lungs posteriorly. The child took no notice of anything, but swallowed when liquid food was placed in the mouth. 6 P. M., temp. 101.5°; pulse 165; resp. 92. The respiration was ascending and descending, but no intermissions, as in the Cheyne-Stokes type, occurred.

26*th*. The child died at 2 A M., being able to swallow five minutes before death; no convulsions occurred. The breathing became very slow a short time before life was extinct.

Sectio cadaveris was made twelve hours after death by Drs. C. K. Mills, I. E. Roberts, and myself.

Rigor mortis was just beginning; skin was rough, wrinkled, and very thin. There was great emaciation, the subcutaneous fat having been entirely absorbed. When the child was one year old it weighed twenty-five pounds, but after death, the age being about two years, its weight was only eleven pounds, although its height was probably greater than that of most children of the same age.

Head.—Circumference twenty inches; transverse arch (from mastoid to mastoid) fourteen inches; right half of arch to sagittal suture six and seven-eighths inches; left half seven and one-eighth inches. The head did not seem greatly enlarged. All the bony sutures had closed externally except at the junction of the coronal and sagittal. The skullcap was so thin and yielding, bending like bonnet-board before the saw, that much care had to be exercised in its removal to prevent injuring the brain or its soft coverings. The dura mater was not more adherent to the bone than is usual in children of one or two years of age, except along the line of the sagittal suture, where it became necessary to sever its connections with the bone by the use of considerable force, aided by the knife. After the skullcap was removed, all the sutures were found to be closed internally. The internal surface of the bony cap was smooth, the bones being very transparent, and measuring in the temporal regions from one-tortieth to one-thirty-secondth of an inch in thickness, and in other situations from one-twelfth to one-eighth of an inch. The dura mater was thin and pale, and its sinuses were nearly empty, containing no clots, and but little blood. The pia mater on the convexity of the brain was so attenuated and transparent that it was difficult to distinguish its pres-

ence. The various fissures, primary and secondary, could be traced without removing this membrane. The pia mater over the orbital surfaces of the frontal, the basal surfaces of the temporal and occipital lobes presented the same attenuated, transparent appearance seen on the convexity. The membrane covering the anterior and posterior sub-arachnoidian spaces on first exposure seemed to be opaque, but on cutting it a serous fluid escaped, leaving it of the usual appearance. A few millimetres to the left of the optic chiasm, two yellowish-white spots about one-eighth of an inch in their greatest dimensions, were seen in the substance of the pia mater.

The convex surfaces of both hemispheres of the brain on palpation gave decided fluctuation. On cutting into the lateral ventricles, they were found filled with a colorless watery fluid. The bodies and horns of these ventricles were enormously dilated. The foramen of Monro, the third and fourth ventricles, and the Sylvian aqueduct were enlarged to about two or three times their normal dimensions. On examining the walls of the lateral ventricles and their much dilated horns, prominent veins could be seen everywhere forming beautiful arborescent appearances. The veins of the intra-ventricular striate bodies, the veins of the choroid plexuses, and the veins of Galen themselves, were enlarged and prominent. The ventricular fluid measured fifteen ounces. Measuring from the anterior extremity of the anterior horn to the posterior extremity of the posterior horn, the greatest length of each lateral ventricle was six and a quarter inches. Both ventricles appeared to be dilated equally. The thickness of the brain substance constituting the walls of the lateral ventricles was measured at several points. It was thinnest in the region bounding the dilated posterior horn, within and below, where it measured about one-sixth of an inch. Sections through the gray and white matters showed no punctæ vasculosæ, the brain-substance throughout being markedly bloodless. It was firm and cut cleanly, not adhering to the knife.

The cervical and upper dorsal portions of the spinal cord and its membranes presented nothing abnormal on macroscopic examination.

Thorax.—No pleuritic adhesions or effusion. The smaller bronchial tubes were nearly filled with muco-pus. The lower and posterior portions of the lungs were œdematous and congested. The apices of the lungs were firmer than normal, and in the right apex a small cicatrix and several little nodules. The heart was very small, but apparently normal. The abdominal organs were not examined.

Parts of the brain and the cervical portion of the spinal cord and their membranes, and a small piece of the apex of the right lung, were properly hardened and sent to Dr. L. Brewer Hall, a practical microscopist, for examination. The results of his examinations he gives as follows:—

"Sections of the spinal cord and membranes show oval masses of rounded cells along the vessels of the dura mater, lying chiefly outside their walls. In some places the coats of the vessels are thickened; the nervous tissue appears normal. Similar appearances are occasionally seen in the membranes of the brain; many sections, however, show nothing abnormal. In the apex of the lung there are minute inflammatory spots, visible in some of the sections only. Parts of these consist of rounded cells about and within a bronchus, but others are surrounding bloodvessels and not encroaching upon their calibres.

"The pathological condition is tubercle, chiefly of the membranes of the spinal cord, less abundant in the meninges of the brain, and rare and small, though present, in the lung."

In the history of the case which I have given in detail to-night, periodicity of temperature, attended

by numerous exaggerated symptoms of brain disturbance, was well marked. During these periods, in which the case ran a less acute course, a slight exacerbation of fever was noticed nearly every alternate day. After the first few weeks of the disease, when the fever began to reach its highest points, the paroxysms of fever, headache, and restlessness varied from a few hours to one or two days. The fever invariably passed off by free perspiration. On some days the fever was of the remittent, and on others of the intermittent type. I have frequently observed a striking similarity between the temperature variations of malarial fevers and those due to brain diseases; but in no instance have I seen the temperature of the latter diseases resemble that of the former when the membranes or cortical substance of the brain was uninvolved by disease. Fevers of cerebral origin differ from those of malarial poisoning, in that the paroxysms of the former are less regular in their occurrence and duration, and are not so easily prevented by large doses of quinine, although in the case reported in this paper, large quantities of quinine given daily seemed to prevent excessive rise of temperature, and apparently lessened the frequency of the paroxysms.

During the first few days of the child's sickness, the diagnosis was between spurious hydrocephalus, acute meningitis, tubercular meningitis, and sporadic cerebro-spinal meningitis; later between the last two and chronic hydrocephalus. A careful study of the morbid anatomy of this case, however, discovers no lesion that is not found in one or more of the above diseases, yet the clinical history, when taken in connection with the varying records given by the body

and head temperatures, shows the disease to be possessed of a certain individuality, which, with a number of carefully studied histories, we might be able to recognize.

The quantities of morphia, two to three grains in twenty-four hours, which were given to a child less than two years old without producing death, seem almost incredible. At first, twenty drops of camphorated tincture of opium were sufficient to quiet the child for a short time, but this preparation of opium was gradually increased until about two ounces were necessary for twenty-four hours. In the same way morphia was increased as toleration was established, the maximum quantity only being reached after regularly giving the drug a number of weeks. After having discontinued the use of morphia two months, the return of head-pain compelled me to again resort to it. This time, also, small doses were at first sufficient to quiet the child, but in the course of a few weeks one-third to one-half grain doses were required. While large doses of opium were being administered, neither diarrhœa nor vomiting was present.

The extraordinary duration of the tubercular disease of the cerebro-spinal membranes, in this case, extending over a period of nearly eight months, simulated rare cases of sporadic cerebro-spinal meningitis, which may, also, after further investigation, be found to be tubercular in their nature. Ordinary cases of tubercular meningitis run their course in from two to four weeks. A few have lasted about ten weeks. There is, however, on record, one rare case of tuberculosis of the cerebral membranes (*St.*

George's Hospital Reports for 1879), in which the head symptoms were of twelve months' duration. The patient was a man thirty-five years old, admitted into the hospital a few hours before his death. The man and his wife stated that the affection had begun suddenly twelve months before, with embarrassment of speech and deglutition. Failure of the sight, with severe cough and wasting were of six months' date. "On admission, the symptoms were general weakness of the limbs, the arms in particular moving with jerking action, and requiring concentration of thought to influence them at all; loss of power of deglutition, during attempts at which the head was turned around to the right; inability to protrude the tongue; complete anæsthesia of the soft palate, indistinctness of speech, the vocal cords seeming partially paralyzed; dulness of vision, ptosis, frowning of the forehead, sluggishness of the pupils and drowsiness; but no derangement of mind." The post-mortem appearances were, "A few tubercles in the arachnoid on the convexity of the brain; a little lymph in the interpeduncular space, without tubercle; the medulla and its nerves normal; left kidney small." The case I have just quoted, when taken in connection with the one reported in this paper, is particularly interesting on account of its long duration, decided motor disturbance of cerebral origin, and the slight lesion found in the arachnoid of the brain, the spinal cord apparently being uninvolved. The long duration of the case I have reported to-night may, I think, have been due to tubercles being very sparsely scattered on the membranes of the brain and cord, without any

other organ becoming sufficiently affected to endanger life.

In cases of tubercular cerebro-spinal meningitis are the paralysis and rigidity of spinal or cerebral origin? H. Rendu (Debove, *Le Progrès Médical* for 1879) says, "I think I am authorized in concluding that the existence of tubercular granulations on the spinal meninges have only a purely anatomical interest, and that their value, in a clinical point of view has been, perhaps, exaggerated."

In Debove's case (*Ibid.*), the chief symptoms were due to the spinal lesions, which were most marked in the dorsal and lumbar regions. And in the one related by Chateaufort (*Ibid.*), the spinal symptoms were first and most prominent. The patient was suffering from advanced pulmonary tuberculosis. A few days before his death he felt a sharp pain in the spine radiating to the lower limbs. Marked rigidity of the trunk was present, and finally delirium and paralysis. In the case of the child attended by me, the lesions of the spinal membranes were greater than those found in the cerebral meninges.

"Rendu (A. McL. Hamilton, *Nervous Diseases*, 1881; *Review in Gaz. des Hôpitaux*, Jan. 15, 1873) affirms that whenever there is paralysis of permanent form there must be some obliteration from fibrinous exudation and consequent softening, and he does not believe that scattered granulations or ventricular effusion are alone sufficient for its causation." Ventricular effusion, as seen in chronic hydrocephalus, especially, is not usually attended by rigidity of the muscles of the extremities. The pathological lesions demonstrated by different observers of tuberculosis

of the nervous centres abundantly prove that paralysis or contractions of a permanent character may result from lesions in the spinal cord or brain, or in both. In my case there was no paralysis, although a large quantity of fluid filled and distended the ventricles, probably because, as contended for by Rendu, no lesions of softening existed. The rigidity in this case may be accounted for by the comparatively sudden ventricular effusion, which apparently took place a few months before the child's death; the brain being thus crippled, its inhibitory influence over the cord was lessened, and the latter nerve centre kept up a constant muscular contraction. What I think is a more plausible explanation of the condition, is found in the presence of scattered tubercle, attended by inflammation, in the membranes of the brain and cord. Tubercles were few in both situations, but most abundant in the membranes of the medulla, the very centre of reflex actions.

The macroscopical appearances of the brain and cord and their membranes were those of great anæmia of the parts. Nothing suggestive of tubercle or even inflammation, save two small yellowish nodules at the left of the optic chiasm, was seen. Many of the microscopical sections, also, showed neither tubercular nor inflammatory lesions. It is evident from a careful study of this case that examinations must be most careful and thorough before giving an opinion in regard to the presence or absence of tubercles or of inflammation of the membranes of the brain and cord.

The pulse frequency of the case reported to-night, like that of tubercular meningitis, divides the disease into three unequal stages. The first stage was

of five months' duration, during which the pulse was very rapid, being, on the average, about 130 to 135 per minute, but frequently reaching 160 to 170, and on a few occasions 180. The second stage was one month long, the pulse ranging from 76 to 120, the average being about 100. The last stage extended over a period of about one and a half months, the pulse being again rather rapid, from 101 to 148, the average being about 130. During the early part of the disease respiration was most rapid when the pulse was most frequent; but later, slow respiration (8 or 10 per minute) was accompanied by rapid pulse (140 to 144).

Irregularity and sudden changes were well shown by the axillary thermometric records. On May 1, the temperature fell from 104° to 95.6°, or 8.4° in twelve hours; and three days later, from 105° to 96.5°, or 8.5° in about the same length of time. Diurnal variations of temperature from 4° to 6° were common. The temperatures in each axilla were compared sixty times; in five of these the left axillary heat exceeded the right from .1° to 1.8°, the average difference being .5°; the temperatures were equal eight times, showing the right axilla to have been warmer than the left forty-seven times, the difference varying from .1° to 1.8°, the average being only .3°. Part of the variation, I think, may be accounted for by the difficulty we encounter in keeping children quiet while registering their temperatures.

Cerebral Thermometry.—From my paper which I read before the College, May 3, 1882 (Report of Three Cases of Abscess of the Brain), I quote the following: "Dr. Gray found the average normal temperatures of

the stations on the side of the head to be for the frontal .65° F., for the parietal .86° F., for the occipital .72° F., higher than those of the corresponding stations on the right side. He gave as the average normal temperature of the right frontal station 93.71° F., of the left 94.36° F.; of the right parietal 93.59° F., of the left 94.44° F.; of the right occipital 91.94° F., of the left 92.66° F. Variations of more than 1.5° he considered suspicious of disease at that point, and of more than 2° strong evidence of a pathological condition."

Dr. J. S. Lombard published in book form, in 1879, the results of upwards of 60,000 observations on the regional temperature of the head made by means of the thermo-electric apparatus. (*Experimental Researches on the Regional Temperature of the Head, etc.*, by J. S. Lombard, M.D.) His averages of temperatures of the different regions of the head are—

Average Temperature.

	Anterior region.	Middle region.	Posterior region.
Right side,	92.942° F.	92.073° F.	92.343° F.
Left side,	92.825	92.114	92.267
Both sides,	92.883	92.093	92.309

He says that "higher values, especially for the anterior, are more frequently met with in ordinary examinations; but it is extremely difficult to arrive at satisfactory conclusions as to average absolute temperature from observations made upon individuals while engaged in the ordinary avocations of life, the results thus obtained being very variable. Under such circumstances, a temperature of 95.36° F. for the anterior region; one of 94.1° F. for the middle region, and one of 93.56° F. for the posterior region,

would probably represent more correctly, in the majority of cases, the highest absolute temperatures."

It will be seen by Lombard's conclusions that the temperatures of the anterior and posterior regions are higher, and that of the middle region lower on the right than on the left side of the head. In a later work by the same author (*Experimental Researches on some points Relating to the Normal Temperature of the Head*, by J. S. Lombard, M.D., 1880), it is stated that—"It was found in these experiments, among the results, that every one of the small divisions of the surface of the head might be hotter on the right side or on the left side in turn, and also that many of them showed at times equality of temperature of the two sides."

In numerous observations made by myself on cerebral temperature of persons in health, and of persons suffering from general febrile conditions, the right side of the head has been found, in many instances, slightly warmer than the left. When surface thermometers are employed to register the heat of the head in disease, I think, if we take the "highest absolute average temperatures" of Lombard for comparison, we shall be less likely to come to erroneous conclusions. If we take, then, the comparatively high temperatures, 95.36° F., 94.1° F., and 93.56° F., for the normal heat of the anterior, middle, and posterior regions of the head respectively, we shall find, when we compare them with the results of the numerous temperature observations made on the patient whose case is reported in this paper, that much of interest, and, probably, of clinical value, may be derived by a careful study of cerebral thermometry in these cases.

The results of the local thermometric records in this case may be thus summed up:—

The surface temperatures of the head and of the upper posterior cervical region were registered on ninety-seven different occasions, during which four hundred and eleven surface temperature observations were made on these parts. The cerebral temperatures exceeded the axillary forty-six times; that of the axilla was higher than the head temperatures forty-seven times; and the temperatures of the head and axilla were equal four times.

Once the temperatures in the axilla and over the upper cervical region were equal; four times the temperature of the latter region fell below that of the axilla, varying from .1° to 1°; twenty-four times the heat of the upper cervical region exceeded that of the axilla, it having been on one occasion as much as 3.4° above it. The temperature in the upper cervical region was usually greater than that of the head, it having been higher nineteen times out of twenty-nine.

Of the forty-six times that the head temperature was greater than that of the axillary, the heat of the latter region was subnormal sixteen, normal eleven, and above normal nineteen times. Of the forty-seven times that axillary temperature exceeded that of the head, the thermometer in the axilla registered subnormal seven, normal ten, and above normal thirty times.

The lowest head temperature found was 95.8°, the axillary heat at that time being 97.8°. The highest head temperature registered was 103.5°, the axillary being 104°. The lowest axillary temperature recorded

was 93.8°, the cerebral temperature, at the same time, standing at 98°. The highest temperature found in the axilla, at the time of registering the head temperatures, was 104°, the surface thermometer on the head at the same time rose to 103.5°. Once the head temperature was 4.2° greater than the axillary, but the heat of the axilla never exceeded that of the head more than 2°.

The average temperatures of the axilla, of the various regions of the head, and of the upper cervical region, are as follows:—

Parietal station.	Rolandic station.	Posterior frontal station.	Superior frontal station.	Occipital station.
Right side 99.24°	Right side 99.27°	Right side 98.95°	Right side 98.65°	Right side 98.54°
Left " 99.24	Left " 99.22	Left " 98.80	Left " 98.75	Left " 98.28

Middle frontal station 98.08°. Middle occipital 98.39°. Upper cervical region 98.41°. (The average temperature of the upper cervical region was slightly above those of the head and axilla, but the table shows a different average, the discrepancy appearing because the temperature was registered a limited number of times over the upper cervical region.) Axillary region 98.89°.

The head temperatures at no time descended to the normal, although the axillary temperature was either normal or subnormal forty-five times out of ninety-seven comparative examinations. The temperatures of each side of the head were nearly equal. Corresponding regions of the two sides rarely varied more than a fraction of a degree Fahr. The parietal stations were the warmest, the middle frontal station, only a little below the middle occipital, was the coolest.

If the results of the cerebral temperature observations of this case are compared with the observations made by Dr. Mary Putnam-Jacobi (*The Journal of*

Mental and Nervous Diseases, Jan. 1880), on a case of suppurative tubercular meningo-cephalitis, a striking difference in the height of the temperature will be found, partially confirming the conclusions that I had almost arrived at from numerous observations on cerebral thermometry, viz., that brain lesions accompanied by congestion or inflammation are attended by higher head temperature before than after suppuration supervenes.

The following conclusions may be drawn from the contents of the paper:—

1. Tubercles occur in the membranes of the brain and cord, but they are more frequently found in the former than in the latter situation.

2. Tubercular deposit may first take place in the meninges of the cord and then extend to those of the brain, although the reverse is the rule.

3. Tuberculosis of the cord, contrary to the views of Rendu, has more than an anatomical interest.

4. Tubercular cerebro-spinal meningitis gives rise to special symptoms, and by a careful analysis of a number of cases, it may be recognized.

5. It is probable that many cases of so-called sporadic cerebro-spinal meningitis, that have a duration of several months and then prove fatal, are tubercular in character.

6. Paralysis or contractions may be due to brain or spinal lesions, or to both.

7. When cerebral fever simulates the periodic manifestations of malaria, the lesion is probably in the membranes or cortical substance of the brain, or in such a position as to exert pressure upon these structures.

8. Ventricular effusion probably does not give rise to paralysis or contractions; the former motor disturbance in these cases being due to softening of motor zones, and the latter to irritation of the meninges of the brain and cord.

9. Repeated microscopical sections may have to be made in certain cases of tuberculosis of the meninges before the nature of the disease is detected.

10. Right or left side of the head may, in turn, be the warmest in health.

11. When surface thermometers are used to register the cerebral temperature in disease, the normal averages should be taken to be 1° to 1.5° higher than those given by Gray and others.

12. The head temperature in disease of the brain may equal or exceed the heat of the axilla for a length of time.

13. In cerebral lesions, the temperature of the head is not marked by those sudden variations manifested by the axillary temperature in these cases.

14. Variations of head temperature in diseases of the brain take place comparatively slowly. The tendency of the heat of the head to remain permanently above the normal, while that of the axilla is normal or several degrees below, is the strongest evidence of organic disease.

15. The thermometer and the microscope in the case reported agreed in locating the greatest inflammatory trouble in the upper cervical portion of the cord.

16. Brain lesions attended by congestion or inflammation have a higher local temperature than suppuration going on within the cranial cavity.

[After the reading of the preceding paper:—]

Dr. Roberts Bartholow said: Since you have called on me, Mr. President, I will make some remarks on the very elaborate report which we have just heard. The impression which the clinical history makes on me is that the case if tubercular is one rather of general tuberculosis than of tubercular cerebro-spinal meningitis. It may be objected to this view that in every case of tubercular meningitis there is more or less generalized tubercular deposit. It seems to me, however, hardly logical to place so much emphasis on the spinal affection. In most cases of unquestionable tubercular meningitis the principal seat of tubercular deposit is at the base, hence the term *basilar meningitis*, parts in immediate connection with the spinal meninges. It is the more philosophical, therefore, it seems to me, to regard such cases as examples of general tuberculosis; now one set of organs, now another set, being the points selected for a special tubercular deposit. I do not, therefore, believe in the existence of any cases of tubercular cerebral meningitis, or spinal meningitis, *per se*, but in a general tuberculosis, with special implication of certain organs. Under such circumstances the symptomatology will vary with the particular functions of organs the most involved.

As regards the cerebral thermometry on which the essayist has much dilated, I beg to say a word or two. Up to the present, cerebral temperature changes have not afforded us much information. As you are well aware, Mr. President, Lombard achieved but trifling results in his study of cerebral temperatures, although he employed the most elaborate instruments of precision now available for the purpose. Indeed it is now well understood that the temperature of the scalp affords no certain indication, if any indication whatever, of the cerebral temperature. A few years ago a physician of New York, Dr. Amidon, received a prize, and attracted no little attention for an essay, in which he attempted to show that certain motor areas in the brain could be mapped out by the temperature changes in the scalp, induced by active exercise of the muscles in anatomical relation with these motor areas. But it was soon ascertained that the temperature changes were limited to the scalp, and that the "willed movements" do not, so far as at present known, so raise the temperature of the motor cortical centres as to change the temperature of the scalp

In fact, it is now known that the temperature of distant parts is influenced by slight excitation of the surface. Thus, Strumpf, of Dusseldorf, has shown that faradic stimulation of the skin of one member affects the heat of another member. There are numerous examples of the same kind. In this way the influence of muscular action on the temperature of the scalp is explained. We cannot, therefore, formulate conclusions in regard to the condition of the brain from observations on the heat of the scalp. The whole subject is still *sub judice*. Hence, I think, it is very unsafe to draw conclusions from cerebral thermometry in the present state of our knowledge.

Dr. DA COSTA remarked that, notwithstanding the elaborate and able report of the case, he had some doubts with reference to its character. The sudden beginning was unlike that of tubercular meningitis, rather that of those instances of cerebro-spinal fever which begin acutely and may become chronic. In support of this view there was an eruption of an ecchymotic kind, in the first twenty-four hours, such as occurs in cerebro-spinal fever; there is no eruption in tubercular meningitis. Again, there was no very positive evidence of tubercle in the lung, and he thought it unlikely that a case of tubercular meningitis should have existed for months, affecting the brain and cord, and no decided tubercles in the lungs be found.

Dr. CHAS. K. MILLS referred to the fact that in a number of cases of tubercular meningitis, some in children and some in adults, some general and some localized, he had observed eruptions differing in character; sometimes erythematous, sometimes vesicular, sometimes ecchymotic, and probably, in the latter case, due to extreme vaso-motor paresis.

He alluded also to four cases of tubercular meningitis seen with Drs. Franklin and Ott. In three of these gross appearances were present; in the fourth the appearances were similar to those exhibited by Dr. Eskridge's patient. No tubercular masses were visible anywhere, either at base or convexity, and yet the microscope revealed miliary tubercles in great abundance. These observations show the importance of not concluding that a case is non-tubercular simply from macroscopic examination. Probably many of the statements of the older observers are not to be relied upon in this respect.

Dr. ESKRIDGE, in closing the discussion, said that it was probably exceedingly rare to have tubercular inflammation of the membranes of the brain or cord without tubercles occurring in other portions of the body. In pulmonary tuberculosis it was common for other organs than the lungs to be attacked. He was inclined to think that if, in certain cases, we follow the custom of locating the disease in one or more organs, those should be selected which were most early and prominently affected. In the case under consideration the early symptoms, and, in fact, those of any prominence at all throughout the disease, were due to the cerebro-spinal lesions. The point was well taken by the first speaker, as the microscope shows that most cases of so-called local tuberculosis are examples of general tubercular affections. The same speaker was also justified in questioning the claims that had been made by some observers for cerebral thermometry. Dr. E. was not yet satisfied that surface thermometers would enable us to locate lesions in different portions of the brain with sufficient accuracy for general diagnostic or therapeutic purposes. He desired to call special attention to one point in connection with his observations on the surface temperature of the head in this and other cases, viz., that a sustained high head temperature extending over a considerable period, while the axillary heat was normal or below, pointed to organic lesion of the brain or its membranes. Again, in brain diseases, the cephalic temperature is less variable than the axillary heat in the same cases. The sudden beginning and long duration of the case, the history of which he had given in detail, and the presence in it of an early eruption, was thought by the second speaker to point to sporadic cerebro-spinal meningitis rather than to the tubercular form of meningeal disease. Cases of tubercular meningitis without prodromata were the exception, but they did occur. One or two of the three cases referred to by Dr. Mills, and reported in a joint paper by himself and Dr. Ott (*Philadelphia Medical and Surgical Reporter*, July 3, 1881), were ushered in by convulsions, the previous health of the children having been good. Dr. Gee (Reynolds's *System of Medicine*) says "that out of twenty-six cases of primary tubercular meningitis (the diagnosis having been confirmed by a *post-mortem* examination in all) there were only two in which premonitory symptoms had not been noticed." The long duration (nearly eight months) of the present case would class it with the curiosities of

medical literature, but it was not without a parallel, for Dr. E. had quoted a case in the early part of his paper, from the *St. George's Hospital Reports* for 1879, in which the meningeal symptoms extended over a period of twelve months. Other cases of tubercular meningitis that have lasted about ten weeks have been reported. The eruption referred to in his case, and thought by some to be of considerable diagnostic value in this particular instance, looked like blood extravasated under the skin, but it disappeared as soon as the child was put in a warm bath, an hour or two after he first saw it. It did not again make its appearance.

The absence of macroscopical appearances of inflammation militated, to his mind, very strongly against the supposition of the disease having been sporadic cerebro-spinal inflammation. So far as he knew, if the patient lived a few days after being seized with the last-named disease, inflammatory deposits were always apparent to the unaided eye. If it be argued that the present case lasted so long that the exudate was absorbed, he would like to know the cause of death. Irritation was increasing, as shown by muscular contractions, etc., during the last two months of the little fellow's illness, yet at the autopsy no visible exudation was present. In rare cases of tubercular meningitis the macroscopical appearances of inflammation have not been well marked. In one of the cases reported by Drs. Mills and Ott no tubercles were seen. In one of tubercular cerebro-spinal meningitis, reported by Galliaux, the "membranes were not thickened or much congested, presenting nearly their normal appearance." At the autopsy of Dr. Eskridge's case the presence of tubercles was not suspected by Dr. Mills or himself, and inflammation, which, during life, was thought to exist, could not be detected by any exudate. Hardened portions of the brain and cord and their membranes, and of the lung, were given to an expert microscopist who knew nothing of the history of the little patient, and he was asked to decide whether inflammation had existed. He found, as stated in his report, tubercles in portions of all the organs examined. If it were not for the microscopic revelations which Dr. Eskridge did not feel inclined to dispute, the case he had reported to the College would have to be considered one of chronic cerebro-spinal meningitis minus appreciable exudation.

TEMPERATURE, PULSE, AND RESPIRATION FROM APRIL 16, 1882, TO NOVEMBER 25, 1882.

April	16,	12 M.	.	.	temperature	103°;		pulse	150;	respiration	84.
"		4 P. M.	.	.	—	—	—	"	180;	"	96.
"		6 P. M.	.	.	"		102.6;	"	180;	"	96.
"		10 P. M.	.	.	—	—	—	"	160;	"	92.
"	17,	8 A. M.	.	.	"		101.5;	"	160;	"	84.
"		12 M.	.	.	—	—	—	"	150;	"	76.
"		7 P. M.	.	.	"		102;	"	140;	"	56.
"	18,	9 A. M.	.	.	"		101;	"	130;	"	38.
"		1 P. M.	.	.	—	—	—	"	140;	"	38.
"		7 P. M.	.	.	"		102;	"	135;	"	44.
"	19,	8 A. M.	.	.	"		100;	"	118;	"	50.
"		2 P. M.	.	.	—	—	—	"	116;	"	50.
"		9 P. M.	.	.	"		103.8;	"	135;	"	84
"	20,	8 A. M	.	.	"		102.6;	"	135;	"	48.
"		2 P. M.	.	.	—	—	—	"	130;	"	48.
"		7 P. M.	.	.	"		102.8;	"	140;	"	60.
"	21,	9 A. M.	.	.	"		101.8;	"	121;	"	42.
"		6 P. M.	.	.	"		102.6;	"	120;	"	50.
"	22,	9 A. M.	.	.	"		103;	"	112;	"	40.
"		3 P. M.	.	.	"		103.5;	"	130;	"	60.
"	23,	11 A. M.	.	.	"		102.5;	"	140;	"	60.
"		8 P. M.	.	.	"		102;	"	136;	"	60.
"	24,	11 A. M.	.	.	"		98;	"	120;	"	48
"		6 P. M.	.	.	"		105;	"	130;	"	60.
"	25,	10 A. M.	.	.	"		98;	"	130;	"	40.
"		6 P. M.	.	.	"		100.2;	"	124;	"	40.
"	26,	9 A. M.	.	.	—	—	—	"	120;	"	60.
"		8 P. M.	.	.	"		98;	"	130;	"	50.
"	27,	9 A. M.	.	.	"		103;	"	150;	"	68.
"		5 P. M.	.	.	"		98;	"	130;	"	64.
"	28,	10 A. M.	.	.	"		98.2;	"	112;	"	62.
"		6 P. M.	.	.	"		98;	"	120;	"	60.
"	29,	9 A. M.	.	.	"		101;	"	130;	"	68.
"		6 P. M.	.	.	"		102;	"	140;	"	70.
"	30,	10 A. M.	.	.	"		98;	"	116;	"	50.
"		4 P. M.	.	.	"		103.5;	"	160;	"	60.
"		10 P. M.	.	.	"		104;	"	170;	"	70.
May	1,	10 A. M.	.	.	"		95.6;	"	120;	"	48.
"		6 P. M.	.	.	"		98;	"	140;	"	60.
"	2,	11 A. M.	.	.	"		103.6;	"	160;	"	60.
"		3 P. M.	.	.	"		102;	"	170;	"	56.
"		8 P. M.	.	.	"		98.5;	"	170;	"	60.
"	3,	9 A. M.	.	.	"		98;	"	160;	"	52.
"		6 P. M.	.	.	"		105;	"	180;	"	60.
"	4,	10 A. M.	.	.	"		96.5;	"	170;	"	60.
"		3 P. M.	.	.	"		98;	"	160;	"	60.
"	5,	9 A. M.	.	.	"		97.2;	"	170;	"	60.
"		6 P. M.	.	.	"		98.4;	"	150;	"	56.
"	6,	9 A. M.	.	.	"		98.4;	"	160;	"	56.

TEMPERATURE, PULSE, AND RESPIRATION—*continued.*

May	6,	5 P. M.	.	.	temperature,	100;	pulse	140;	respiration	48.
"	7,	9 A. M.	.	.	"	105.2;	"	152;	"	72.
"		6 P. M.	.	.	"	103;	"	160;	"	52.
"	8,	11 A. M.	.	.	"	100.8;	"	130;	"	50.
"		6 P. M.	.	.	"	99.6;	"	140;	"	48.
"	9,	9 A. M.	.	.	"	104;	"	144;	"	76.
"		6 P. M.	.	.	"	100.8;	"	136;	"	56.
"	10,	9 A. M.	.	.	"	105.6;	"	144;	"	72.
"		6 P. M.	temp.	{ right axillary		101.8; }	"	144;	"	76.
				{ left	"	101.6; }				
"	11,	10 A. M.	"	{ right	"	105.5; }	"	154;	"	64.
				{ left	"	105.2; }				
"		6 P. M.	"	{ right	"	104.9; }	"	180;	"	84.
				{ left	"	105; }				
"	12,	10 A. M.	"	{ right	"	98.2; }	"	152;	"	62.
				{ left	"	98; }				
"		6 P. M.	.	.	temperature	99;	"	160;	"	60.
"	13,	9 A. M.	.	.	"	99;	"	160;	"	64.
"		6 P. M.	.	.	"	104;	"	176;	"	92.
"	14,	10 A. M.	.	.	"	98.6;	"	160;	"	56.
"		5 P. M.	.	.	"	99;	"	162;	"	86.
"	15,	9 A. M.	.	.	"	97.5;	"	160;	"	84.
"		7 P. M.	.	.	"	98;	"	140;	"	72.
"	16,	9 A. M.	.	.	"	97.8;	"	154;	"	60.
"		6 P. M.	.	.	"	98;	"	160;	"	52.
"	17,	9 A. M.	.	.	"	97;	"	140;	"	64.
"		6 P. M.	.	.	"	98;	"	132;	"	52.
"	18,	10 A. M.	temp.	{ right axillary		101; }	"	170;	"	68.
				{ left	"	100.4; }				
"		6 P. M.	"	{ right	"	100.4; }	"	170;	"	72.
				{ left	"	99.4; }				
"	19,	11 A. M.	"	{ right	"	98.9; }	"	124;	"	52.
				{ left	"	97.8; }				
"		8 P. M.	"	{ right	"	101.4; }	"	126;	"	68.
				{ left	"	100.6; }				
"	20,	9 A. M.	"	{ right	"	98.8; }	"	128;	"	36.
				{ left	"	99.1; }				
"		7 P. M.	.	.	temperature	97.8;	"	136;	"	44.
"	21,	10 A. M.	temp.	{ right axillary		103.9; }	"	160;	"	72.
				{ left	"	103.8; }				
"		4 P. M.	"	{ right	"	99; }	"	124;	"	56.
				{ left	"	98.8; }				
"	22,	10 A. M.	"	{ right	"	99 2; }	"	135;	"	52.
				{ left	"	101; }				
"		7 P. M.	"	{ right	"	99 6; }	"	116;	"	54.
				{ left	"	100.2; }				
"	23,	10 A. M.	"	{ right	"	100; }	"	138;	"	84.
				{ left	"	100; }				
"		7 P. M.	.	.	temperature	103.5;	"	130;	"	60.
"	24,	9 A. M.	temp.	{ right axillary		100; }	"	140;	"	60.
				{ left	"	99; }				
"		7 P. M.	"	{ right	"	98.5; }	"	148;	"	64.
				{ left	"	97.2; }				
"	25,	8 A. M.	"	{ right	"	103.6; }	"	148;	"	72.
				{ left	"	103; }				

TEMPERATURE, PULSE, AND RESPIRATION—*continued.*

May 25,	6 P. M.	temp.	right axillary	97.8;	pulse	124;	respiration	40.
			left	"	97.8;				
" 26,	9 A. M.	"	right	"	103;	"	146;	"	80.
			left	"	103;				
"	6 P. M.	"	right	"	98.4;	"	120;	"	60.
			left	"	97.8;				
" 27,	9 A. M.	"	right	"	102.8;	"	144;	"	52.
			left	"	102.1;				
"	6 P. M.	"	right	"	99.7;	"	124;	"	52.
			left	"	99.1;				
" 28,	10 A. M.	"	right	"	101.4;	"	168;	"	68.
			left	"	99.9;				
"	7 P. M.	"	right	"	102;	"	144;	"	52.
			left	"	101.8;				
" 29,	10 A. M.	"	right	"	101.5;	"	134;	"	62.
			left	"	101.1;				
"	6 P. M.	"	right	"	99.7;	"	134;	"	58.
			left	"	99.7;				
" 30,	10 A. M.	"	right	"	101.5;	"	130;	"	50.
			left	"	100.9;				
"	6 P. M.	"	right	"	101;	"	140;	"	72.
			left	"	101;				
" 31,	9 A. M.	"	right	"	104.2;	"	130;	"	68.
			left	"	103.4;				
"	8 P. M.	"	right	"	100.3;	"	144;	"	64.
			left	"	99.2;				
June 1,	10 A. M.	"	right	"	97.6;	"	140;	"	54.
			left	"	97.3;				
"	6 P. M.	"	right	"	97.4;	"	128;	"	62.
			left	"	96.9;				
" 2,	10 A. M.	"	right	"	99.5;	"	140;	"	48.
			left	"	98.6;				
" 3,	10 A. M.	"	right	"	102.2;	"	164;	"	76.
			left	"	101.7;				
"	6 P. M.	"	right	"	100;	"	124;	"	52.
			left	"	99.5;				
" 4,	12 M.	"	right	"	97.2;	"	112;	"	28.
			left	"	97;				
"	9 P. M.	"	right	"	99.4;	"	130;	"	50.
			left	"	98.8;				
" 5,	11 A. M.	"	right	"	99.6;	"	144;	"	28.
			left	"	98.5;				
"	6 P. M.	"	right	"	99.7;	"	140;	"	60.
			left	"	98.5;				
" 6,	10 A. M.	"	right	"	99.7;	"	130;	"	44.
			left	"	98.6;				
"	6 P. M.	"	right	"	100.2;	"	140;	"	52.
			left	"	98.2;				
" 7,	10 A. M.	"	right	"	103;	"	152;	"	58.
			left	"	102.8;				
"	7 P. M.	"	right	"	98.6;	"	130;	"	52.
			left	"	98.4;				
" 8,	11 A. M.	"	right	"	99.3;	"	140;	"	60.
			left	"	98.7;				
"	7 P. M.	.	.	temperature	99.2;	"	120;	"	40.

TEMPERATURE, PULSE, AND RESPIRATION—*continued.*

June	9,	10 A. M.	temp.	right axillary	103.6;	pulse	140;	respiration	38
"		6 P. M.	"	left "	103.6;	"	180;	"	54.
				right "	104.1;				
				left "	104:				
"	10,	9 A. M	"	right "	98.4;	"	140;	"	48.
				left "	97.7;				
"		6 P. M.	"	right "	97.2;	"	168;	"	50.
				left "	97:				
"	11.	9 A. M.	"	right "	99.4;	"	128;	"	50.
				left "	99.1;				
"		7 P. M.	"	right "	97.8;	"	140;	"	52.
				left "	97.5;				
"	12,	10 A. M.	"	right "	99.3;	"	150;	"	41.
				left "	98.9;				
"		6 P. M.	"	right "	101.8;	"	180;	"	52.
				left "	101.7;				
"	13,	9 A. M.	"	right "	98.2;	"	144;	"	32.
				left "	97.7;				
"	14.	5 P. M.	"	right "	99.3;	"	160;	"	52.
				left "	98.9;				
"	15,	9 A. M.	"	right "	99;	"	120;	"	40.
				left "	99.2;				
"	16.	4 P. M.	"	right "	98.4;	"	140;	"	36.
				left "	98.3;				
"	17.	10 A. M.	.	. temperature	99:	"	160;	"	44.
"	18.	11 A. M.	temp.	right axillary	99.2;	"	130;	"	44.
				left "	99;				
"	19.	12 M.	"	right "	103.4;	"	160;	"	48.
				left "	102.8;				
"	21,	4 P. M.	"	right "	102:	"	160;	"	46.
				left "	101.8;				
"	25,	11 A. M.	"	right "	99;	"	130;	"	40.
				left "	99;				
"	27,	11 A. M.	.	. temperature	98:	"	128;	"	36.
July	1,	8 P. M.	.	. "	97.5;	"	104;	"	34.
"	5,	11 A. M.	.	. "	97.5;	"	104;	"	26.
"	8,	4 P. M.	.	. "	98.6;	"	86;	"	32.
"	9,	10 A. M.	.	. "	98;	"	128;	"	32.
"	10,	10 A. M.	.	. "	97.8;	"	128;	"	24.
"	12.	10 A. M.	.	. "	98.7;	"	140;	"	36.
"	14,	11 A. M.	.	. "	98.9;	"	120;	"	32.
"	16,	6 P. M.	.	. "	99;	"	140;	"	36.
"	19,	4 P. M.	.	. "	100.4;	"	154;	"	40.
"	21,	6 P. M.	.	. "	98.6;	"	120;	"	36.
"	23,	4 P. M.	.	. "	99.4;	"	148;	"	32.
"	26,	11 A. M.	.	. "	99.4;	"	132;	"	48.
"	30,	10 A. M.	.	. "	100.2;	"	120;	"	32.
Aug.	2,	10 A. M.	.	. "	97.9;	"	162;	"	32.
"	6,	10 A. M.	.	. "	99.5;	"	128;	"	30.
"	10,	10 A. M.	.	. "	98.5;	"	148;	"	36.
"	13,	10 A. M.	.	. "	99;	"	132;	"	32.
"	16,	11 A. M.	.	. "	98.8;	"	116;	"	32.
"	17,	6 P. M.	.	. "	99.8;	"	120;	"	32.
"	19,	5 P. M.	.	. "	100.7;	"	116;	"	30.
"	20,	10 A. M.	.	. "	98.6;	"	110;	"	22.

TEMPERATURE, PULSE, AND RESPIRATION—*continued.*

Aug.	21,	7 P. M.	.	.	temperature	99;	pulse	130;	respiration	28.
"	22,	6 P. M.	.	.	"	100;	"	110;	"	28.
"	23,	6 P. M.	.	.	"	101.3;	"	140;	"	40.
"	24,	10 A. M.	.	.	"	101.2;	"	150;	"	20.
"	25,	6 P. M.	.	.	"	102.5;	"	156;	"	50.
"	26,	10 A. M.	.	.	"	101.1;	"	144;	"	21.
"		6 P. M.	.	.	"	100.5;	"	130;	"	28.
"	27,	10 A. M.	.	.	"	98.8;	"	150;	"	60.
"		8 P. M.	.	.	"	98.6;	"	120;	"	22.
"	28,	10 A. M.	.	.	"	100;	"	154;	"	28.
"	29,	10 A. M.	.	.	"	99;	"	120;	"	24.
"	30,	12 M.	.	.	"	99;	"	132;	"	24.
"	31,	11 A. M.	.	.	"	99.9;	"	130;	"	18.
"		9 P. M.	.	.	"	100.4;	"	140;	"	26.
Sept.	1,	9 A. M.	.	.	"	98.6;	"	130;	"	32.
"	2,	9 P. M.	.	.	"	98.4;	"	120;	"	24.
"	3,	4 P. M.	.	.	"	98.8;	"	120;	"	20.
"	4,	10 A. M.	.	.	"	103;	"	140;	"	15.
"		6 P. M.	.	.	"	104;	"	144;	"	10.
"		10 P. M.	.	.	"	103.5;	"	140;	"	8.
"	5,	10 A. M.	.	.	"	102.5;	"	144;	"	28.
"		2 P. M.	.	.	"	103;	"	148;	"	34.
"		6 P. M.	.	.	"	102.6;	"	144;	"	30.
"	6,	10 A. M.	.	.	"	99.4;	"	124;	"	15.
"		5 P. M.	.	.	"	100.6;	"	128;	"	22.
"	7,	9 A. M.	.	.	"	96.8;	"	128;	"	24.
"	8,	10 A. M.	.	.	"	99.8;	"	130;	"	42.
"		6 P. M.	.	.	"	100.4;	"	140;	"	54.
"	9,	10 A. M.	.	.	"	99.3;	"	132;	"	38.
"		6 P. M.	.	.	"	98.9;	"	136;	"	34.
"	10,	9 A. M.	.	.	"	98.1;	"	120;	"	16.
"		5 P. M.	.	.	"	98.8;	"	128;	"	30.
"	11,	10 A. M.	.	.	"	99.2;	"	124;	"	30.
"		6 P. M.	.	.	"	98.4;	"	124;	"	28.
"	12,	7 P. M.	.	.	"	97;	"	112;	"	24.
"	13,	10 A. M.	.	.	"	93.8;	"	96;	"	14.
"		1 P. M.	.	.	"	98.4;	"	112;	"	22.
"	14,	10 A. M.	.	.	"	97.9;	"	112;	"	20.
"	15,	10 A. M.	.	.	"	96.4;	"	96;	"	14.
"	16,	10 A. M.	.	.	"	97.3;	"	96;	"	16.
"		6 P. M.	.	.	"	97.8;	"	110;	"	18.
"	17,	10 A. M.	.	.	"	98.6;	"	108;	"	14.
"		6 P. M.	.	.	"	99.4;	"	106;	"	18.
"	18,	10 A. M.	.	.	"	98.4;	"	100;	"	13.
"		6 P. M.	.	.	"	98.8;	"	106;	"	18.
"	19,	10 A. M.	.	.	"	99.4;	"	118;	"	24.
"		6 P. M.	.	.	"	99;	"	100;	"	24.
"	21,	9 A. M.	.	.	"	96.4;	"	96;	"	20.
"	22,	5 P. M.	.	.	"	98.4;	"	96;	"	20.
"	23,	10 A. M.	.	.	"	98.7;	"	112;	"	24.
"		10 P. M.	.	.	"	98;	"	100;	"	20.
"	24,	4 P. M.	.	.	"	97.8;	"	92;	"	16.
"	25,	10 A. M.	.	.	"	98.1;	"	82;	"	20.
"	26,	10 A. M.	.	.	"	97;	"	88;	"	19.
"	27,	10 A. M.	.	.	"	95.8;	"	88;	"	15.

TEMPERATURE, PULSE, AND RESPIRATION—*continued.*

Sept.	28,	10	A. M.	.	.	temperature	98.8;	pulse	92;	respiration 12.
"	29,	9	A. M.	.	.	"	97.8;	"	76;	" 18.
"	30,	6	P. M.	.	.	"	98.6;	"	116;	" 22.
Oct.	3,	6	P. M.	.	.	"	98 8;	"	108;	" 20.
"	5,	10	P. M.	.	.	"	99;	"	110;	" 20.
"	6,	1	P. M.	.	.	"	100.8;	"	124;	" 24.
"	7,	9	A. M.	.	.	"	100.6;	"	148;	" 24.
"	8,	11	A. M.	.	.	"	100.1;	"	132;	" 28.
"	9,	1	P. M.	.	.	"	100;	"	124;	" 28.
"	10,	1	P. M.	.	.	"	99.8;	"	136;	" 20.
"		9	P. M.	.	.	"	100;	"	116;	" 10.
"	11,	9	A. M.	.	.	"	98.4;	"	104;	" 15.
"	12.	11	A. M.	.	.	"	98;	"	112;	" 24.
"	13,	1	P. M.	.	.	"	101.2;	"	132;	" 30.
"	16,	1	P. M.	.	.	"	101.2;	"	120;	" 32.
"	17,	10	A. M.	.	.	"	99.6;	"	124;	" 24.
"	18,	6	P. M.	.	.	"	99.8;	"	124;	" 32.
"	19,	5	P. M.	.	.	"	100.4;	"	148;	" 30.
"	20,	6	P. M.	.	.	"	99.4;	"	132;	" 56.
"	21,	10	A. M.	.	.	"	100.1;	"	130;	" 24.
"	22,	4	P. M.	.	.	"	97.8;	"	112;	" 24.
"	23,	5	P. M.	.	.	"	99.4;	"	108;	" 22.
"	24,	6	P. M.	.	.	"	100;	"	132;	" 24.
"	25,	5	P. M.	.	.	"	99.8;	"	110;	" 28.
"	26,	6	P. M.	.	.	"	100;	"	144;	" 32.
"	27,	6	P. M.	.	.	"	99;	"	140;	" 44.
"	28.	10	A. M.	.	.	"	100.4;	"	130;	" 10.
"	29,	10	A. M.	.	.	"	99.1;	"	116;	" 14.
"	30,	5	P. M.	.	.	"	100;	"	124;	" 28.
Nov.	3,	1	P. M.	.	.	"	98.6;	"	110;	" 14.
"	11,	5	P. M.	.	.	"	99;	"	110;	" 24.
"	23,	1	P. M.	.	.	"	98;	"	108;	" 28.
"	24,	10	A. M.	.	.	"	96.7;	"	130;	" 40.
"		6	P. M.	.	.	"	99.6;	"	140;	" 52.
"	25,	10	P. M.	.	.	"	101;	"	160;	" 82.
"		6	P. M.	.	.	"	101.5;	"	165;	" 92.

TEMPERATURE AT DIFFERENT CEREBRAL STATIONS FROM JULY 1 TO NOVEMBER 3.

	Right parietal station.	Left parietal station.	Right Rolandic station	Left Rolandic station.	Right posterior frontal station.	Left posterior frontal station.	Right superior frontal station.	Left superior frontal station.	Right occipital station.	Left occipital station	Middle frontal station	Middle occipital station	Upper cervical station	Axillary station.	Middle dorsal station.
July 1, P. M.	97.6°	97.6°	...	...	...	...	...	...	...	...	...	...	...	97.5°	...
" 5, A. M.	...	...	96.8°	97°	...	...	...	...	...	...	...	...	...	97.5	...
" 8, P. M.	98.9	99	...	...	...	...	...	...	...	...	...	...	...	98.1	...
" 10, A. M.	99.4	99.4	...	...	...	...	...	...	...	...	...	...	...	97.8	...
" 12, A. M.	100 9	100.9	...	...	...	...	...	...	...	...	...	...	...	98.7	...
" 16, P. M.	99.2	99.1	...	...	...	...	...	...	...	...	...	...	...	99	...
" 19, P. M.	100.7	100.8	...	...	...	...	...	...	...	...	...	...	...	100.4	...
" 21, P. M.	101	100.8	...	...	...	...	...	...	...	...	...	...	...	98.6	...
" 23, P. M.	100.5	100.6	...	...	...	...	...	...	...	...	...	...	...	99.4	...
" 26, A. M.	99.2	99.8	...	...	...	...	...	...	...	...	...	...	...	99.4	...
Aug. 2, A. M.	98.6	98.7	...	...	...	...	...	...	...	...	...	...	...	97.7	...
" 13, A. M.	100.8	100.6	...	...	...	...	...	...	...	...	...	...	...	99	...
" 16, A. M.	99.6	99.5	...	...	...	...	...	...	...	...	...	...	...	98.4	...
" 17, P. M.	100.6	100.7	...	...	...	...	...	...	...	...	...	...	...	99.8	...
" 19, P. M.	101	100.1	...	...	...	...	...	...	...	...	...	...	...	100.7	...
" 20, P. M.	99	99	...	...	...	...	...	...	...	...	...	...	...	99	...
" 22, P. M.	99.7	99.8	...	...	...	...	...	...	...	...	...	...	...	100	...
" 23, P. M.	100.4	100.5	...	...	...	...	...	...	...	...	...	...	...	101.3	...
" 24, A. M.	101	100.9	...	...	...	...	...	...	...	...	...	...	...	101.2	...
" P. M.	99.2	99.3	...	...	...	...	...	...	...	...	...	...	...	99	...
" 25, P. M.	101.5	101.4	...	...	...	...	...	...	...	...	...	...	...	102.5	...
" 26, A. M.	100.6	100.8	...	...	...	...	...	...	...	...	...	...	...	101.1	...
" P. M.	99.7	99.7	...	...	...	...	...	...	...	...	...	...	...	101.1	...
" 27, A. M.	98.2	98.2	...	...	...	...	...	...	...	...	...	...	...	98.8	...
" P. M.	99	98.9	...	...	...	...	...	...	...	...	...	...	...	98.6	...

TEMPERATURE AT DIFFERENT CEREBRAL STATIONS—*continued*.

	Right parietal station	Left parietal station	Right Rolandic station.	Left Rolandic station.	Right posterior frontal station	Left posterior frontal station	Right superior frontal station.	Left superior frontal station	Right occipital station	Left occipital station	Middle frontal station	Middle occipital station	Parietal station	Ascending station	Middle frontal station.
Aug. 28, A. M.	100.6°	100.6°	...	...	...	...	...	...	...	...	100°	100°	...	100°	...
" P. M.	98.5	98.6	...	...	...	...	...	...	...	...	...	...	...	98.8	...
" 29, A. M.	98.9	98.9	...	...	...	...	...	...	...	...	...	...	...	99	...
" 30, M.	99.3	99.4	...	...	...	...	...	...	...	...	...	...	...	99	...
" 31, A. M.	100	100	...	...	...	...	...	...	...	...	99.5	99.6	...	99.9	...
" P. M.	99.2	99	...	...	98.6°	98.4°	...	...	...	...	98.6	98.7	...	100.4	...
Sept. 1, A. M.	98.5	98.7	98.4°	98.5°	...	...	...	...	...	...	98.4	98.3	...	98.6	...
" 2, P. M.	97.9	97.9	...	...	...	...	...	...	...	...	97.2	97.3	...	98.4	...
" 3, P. M.	99.6	99.5	...	...	...	...	...	...	...	...	99	99.6	...	98.8	...
" 4, A. M.	102	102	...	...	...	...	...	...	...	...	...	...	...	103	...
" M.	103	102.9	...	...	...	...	...	...	...	...	...	...	...	103.5	...
" P. M.	103.5	103.4	...	...	...	...	...	...	...	...	...	...	...	104	...
" 5, A. M.	102.6	102.7	...	...	...	...	...	...	...	...	...	...	...	102.5	...
" P. M.	102	102.1	101.8	101.9	...	...	...	...	...	...	...	...	...	103	...
" P. M.	100.6	100.5	100.4	100.3	...	...	...	...	...	...	...	...	...	102.6	...
" 6, A. M.	100.6	100.7	100.3	100.2	...	...	...	...	...	...	...	...	...	99.4	...
" P. M.	100.6	100.3	100.2	100.2	...	...	...	...	...	...	...	...	...	100.6	...
" 7, A. M.	97	97	97.1	97.2	...	...	...	...	...	...	...	...	...	96.8	...
" P. M.	98	98.1	97.8	98.9	...	...	...	...	...	...	...	...	...	97.6	...
" 8, A. M.	99.6	99.6	99.7	99.8	...	...	...	...	...	...	...	...	...	99.8	...
" P. M.	100.5	100.4	100.4	100.4	...	...	...	...	...	...	...	...	101.2°	100.4	...
" 9, A. M.	99.3	99.2	99.2	99.2	...	...	...	...	...	...	99.2	99.3	99.8	99.3	...
" P. M.	98.9	99	98.6	98.6	...	...	...	...	...	...	...	...	99.2	98.9	...
" 10, A. M.	98.6	98.6	98.5	98.7	...	...	...	...	...	...	98.2	98.5	98.8	98.1	...
" P. M.	98.9	98.7	98.7	98.5	...	...	...	...	...	...	98.9	99.4	98.9	98.8	...

TEMPERATURE AT DIFFERENT CEREBRAL STATIONS—*continued*.

	Right parietal station.	Left parietal station.	Right Rolandic station.	Left Rolandic station.	Right posterior frontal station.	Left posterior frontal station.	Right superior frontal station.	Left superior frontal station.	Right occipital station.	Left occipital station.	Middle frontal station.	Middle occipital station.	Upper cervical station.	Auxiliary station.	Middle dorsal station.
Sept. 11, A. M.	98.9°	97.8°	98.6°	98.6°	...	...	...	...	...	...	97.7°	97.8°	99.2°	99.2°	...
" P. M.	98.1	98	98	97.8	...	...	...	...	...	...	...	...	98.8	98.4	...
" 12, P. M.	97.9	98	97.8	97.9	...	...	...	...	...	...	...	...	98.2	97	...
" 13, A. M.	98	97.8	97.6	97.6	...	...	...	...	...	...	97.6	97.7	97	93.8	...
" P. M.	98.2	98.1	97.9	97.8	...	...	...	...	...	...	97.2	97.9	...	98.4	...
" 14, A. M.	97.7	97.8	97.5	97.6	...	...	...	...	...	...	...	...	98	97.9	...
" 15, A. M.	98.1	98	98	97.9	...	...	...	...	...	...	...	...	97.4	96.4	...
" 16, A. M.	97.6	97.7	97.5	97.7	...	...	...	...	...	...	97.4	97.5	98.8	97.3	95.6°
" P. M.	98.6	98.4	98.9	98.9	...	...	...	...	...	...	...	...	97.9	97.8	...
" 17, A. M.	99	99	99.2	99.1	...	...	...	...	...	...	...	...	99.2	98.4	...
" P. M.	99.6	99.8	99.5	99.6	...	...	...	...	...	...	...	...	100	99.4	...
" 18, A. M.	98.6	98.8	98.5	98.6	...	...	...	...	...	...	...	...	99.4	98.4	...
" P. M.	99	99.1	98.2	98.3	...	...	...	...	...	...	...	...	98.7	98.8	...
" 19, A. M.	99.6	99.7	99.4	99.7	...	...	...	...	...	...	...	...	99.8	99.4	...
" P. M.	99.1	99.2	99.3	99.6	...	...	...	...	...	...	...	...	98.8	99	...
" 21, A. M.	98.8	98.9	98.9	98.9	...	...	...	...	...	...	...	...	98.7	96.4	...
" 22, P. M.	98.5	98.6	98.8	98.7	...	...	...	...	...	...	99	98	99.1	98.4	...
" 23, A. M.	98.7	98.7	98.8	98.9	...	...	...	...	...	...	98.2	98.9	98.8	98.7	...
" P. M.	97.6	97.7	96.5	96.6	...	...	...	...	...	...	97.6	97.6	...	98	...
" 24, P. M.	97.6	97.7	96.6	96.8	...	...	...	...	...	...	95.8	98	99	97.8	...
" 25, A. M.	97.8	97.7	96.7	96.6	...	...	...	...	...	...	...	...	99	98.1	...
" 26, A. M.	98	97.9	97.9	97.6	...	...	...	...	97.5°	97.6°	97	97.4	98.5	97	...
" 27, A. M.	97.6	97.7	97	97	...	...	...	...	...	...	97.6	97.3	99.2	95.8	...
" 28, A. M.	97.6	97.8	96.8	97.1	...	...	...	...	...	...	96.6	98	99	98.8	...
" 29, A. M.	97	97.2	95.8	95.9	...	...	...	...	...	...	...	...	96.8	97.8	...
" 30, P. M.	98.2	98.3	98	98	...	...	...	...	97.9	97.8	...	...	97.8	98.6	...

TEMPERATURE AT DIFFERENT CEREBRAL STATIONS—*continued*.

	Right parietal station.	Left parietal station.	Right Rolandic station.	Left Rolandic station.	Right posterior frontal station	Left posterior frontal station	Right superior frontal station	Left superior frontal station	Right [illegible] station	Left [illegible] station	Middle [illegible] station	Middle [illegible] station	Upper cervical station.	Axillary station.	Middle dorsal station.
Oct. 3, P. M.	98.2°	98.5°	98.1°	98.3°	...	...	...	...	98.1°	98.5°	98.1°	98.7°	...	98.8°	...
" 5, P. M.	98.3	98.2	98.2	98.1	...	...	...	...	98.4	98.1	...	...	...	99	...
" 6, P. M.	100.3	100.4	100.6	100.6	...	...	...	...	...	...	...	...	...	100.8	...
" 7, A. M.	99.7	99.6	100	99.9	98.7°	98.6°	...	...	99.1	99	...	...	...	100.6	...
" 8, A. M.	99.5	99.6	99.6	98.6	...	...	...	...	...	...	...	...	...	100.1	...
" 10, P. M.	...	...	99	98.6	...	...	...	...	...	...	...	...	...	99.8	...
" P. M.	99.4	99.2	99.2	99.3	...	...	...	...	...	...	99	99.1	...	100	...
" 11, A. M.	98.2	98	98	98.1	...	...	...	...	...	...	...	...	...	98.4	...
" 12, A. M.	97.8	97.9	97.6	97.7	...	...	...	...	...	...	...	...	...	98	...
" 13, P. M.	99.2	99	98.6	98.7	...	...	...	...	...	...	...	...	...	101.2	...
" 16, P. M.	100.	99.8	100.5	99.9	...	...	...	...	...	...	...	...	...	101.2	...
" 17, A. M.	98.7	98.4	98.6	98.4	...	...	...	...	...	...	...	...	...	99.6	...
" 18, P. M.	98.5	98.4	98.6	98.4	...	...	98.5°	98.6°	...	...	...	...	...	99.8	...
" 19, P. M.	99.2	99.4	100.1	100.2	99.3	99.2	98.8	98.9	99.2	99.3	...	...	...	100.4	...
" 21, A. M.	99.3	99.4	99	99	...	...	...	...	...	...	...	...	...	100.4	...
" 23, P. M.	98.1	98.2	97	97.1	...	...	...	...	...	...	...	...	...	99.4	...
" 24, P. M.	98.6	98.8	98.4	98.5	...	...	...	...	...	...	...	...	...	100	...
" 26, P. M.	98.7	98.9	97.9	98	...	...	...	...	...	...	...	...	...	100	...
" 28, A. M.	99.3	99.2	98.8	98.6	99.2	99	...	...	99.6	99.7	...	...	...	100.4	...
" 30, P. M.	98.7	98.3	98.3	98.3	...	...	...	...	...	...	...	...	...	100	...
Nov. 3, P. M.	98.4	98.3	98.2	98.1	...	...	...	...	...	...	...	...	...	98.6	...
Total average	99.24°	99.24°	99.21°	99.22°	98.95°	98.80°	98.65°	98.75°	98.54°	98.28°	98.08°	98.39°	98.45°	98.89°	95.6°

DOES EXCISION OF THE LARYNX TEND TO THE PROLONGATION OF LIFE?

By

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[Read April 4, 1883.]

That complete laryngectomy can be performed without sacrifice of life, there is now accumulative evidence.

That every operation involving the removal of the larynx imperils the immediate existence of the patient, the records of the procedure abundantly attest.

That a very large proportion of the subjects operated upon, say nearly fifty per cent. of them, succumb within so brief a period that their deaths may be justly attributed to the operation, an examination into its statistics renders only too obvious.

The writer owns to a prejudice against the operation; he has never witnessed a laryngectomy, nor seen a patient upon whom it had been performed. It may seem rather an assumption on his part, therefore, to criticize an operation of the technical procedure and after-management of which he is practically ignorant.

Let us consult the statistics of the operation, and contrast the average life of those who have been

subjected to it with the average life of patients in similar condition intrusted to palliative treatment.

I have before me, as I write, more or less detailed records of sixty-five operations of complete "extirpation of the larynx," as it is termed.

There are a number of partial excisions, which have not been brought into the subjoined table because the partial operation is not included in the scope of this paper.

TABLE I.—LIST OF COMPLETE LARYNGECTOMIES, INCLUDING AND SUPPLEMENTING THE TABLES OF MACKENZIE, FOULIS, BLUM, AND BUROW.

No.	Operator.	Date.	Age.	Sex.	Disease.	Parts removed.	Result.	Reference.	Remarks.
1	Watson, Patrick Heron (of Edinburgh).	1866	36	M.	Stenosis from syphilis.	Larynx and one ring of trachea.	Death in 3 weeks from pneumonia.	Foulis; Transactions International Medical Congress, Aug. 1881, vol. iii. p. 255.	Pneumonia was suspected before the operation.
2	Billroth (Vienna).	1873 Dec. 31	36	M.	Carcinoma of the larynx.	Larynx, lower third of epiglottis, part of the upper two rings of the trachea.	Death from recurrence 7 months after the operation.	Archiv f. klin. Chirurgie, Bd. xvii. H. ii. p. 343.	Recurrence noted at end of four months.
3	Heine (Prague).	1874 Apl. 28	50	M.	Carcinoma of the larynx.	Entire larynx.	Death from recurrence 6 months after operation.	Arch. f. klin. Chir., Bd. xix. p. 584; Böhm Corresp. Bl. 1874.	
4	Schmidt, M. (Frankfort).	1874 Aug. 12	56	M.	Epithelioma of the larynx.	Thyroid, cricoid, and both arytenoid cartilages.	Death on the fourth day from collapse.	Arch. f. klin. Chir., Bd. xviii. H. i. p. 189.	
5	Maas (Breslau).	1874 June 1	57	M.	"Adeno-fibroma carcinomatosum."	Entire larynx.	Death from pneumonia 2 weeks after operation. "On fourth day," *Blum.*	Arch. f. klin. Chir., Bd. xix. p. 507.	
6	Watson, P. H. (Edinburgh).	1874	60	M.	Epithelioma of larynx extending to left vocal band.	Larynx.	Death from pneumonia in 2 weeks.	Foulis, Trans. Int. Medical Congress, 1881.	
7	Schönborn (Königsberg).	1875 Jan. 22	72	M.	Carcinoma of the larynx.	Entire larynx.	Death on the fourth day.	Berliner klin. Woch., Sept. 20, 1875, p. 525.	
8	Bottini (Turin).	1875 Feb. 6	24	M.	Sarcoma (partly round-celled, partly spindle-celled) of the larynx.	Entire larynx.	WELL APRIL 19, 1881, probably still alive.	Communicazione letta Junanzi, la R. Academia di Medicina di Torino, April 30, 1875. Letter from Prof. Bottini to Dr. Foulis.	The operation entailed copious hemorrhage, and was followed by severe erysipelas.

TABLE I—*continued.*

No.	Operator.	Date.	Age.	Sex.	Disease.	Parts removed.	Result	Reference	Remarks.
9	Langenbeck (Berlin).	1875 July 21	57	M.	Carcinoma of upper part of larynx, of the epiglottis, and of the hyoid bone.	Entire larynx, hyoid bone, part of the tongue, pharynx, and œsophagus.	Death from recurrence in cervical lymphatic glands, 4 months after.	Berlin. klin. Woch., 1875, No. 33, p. 453; Arch. f. klin. Chir., Bd. xxi. Supplem. p. 136.	
10	Multanowski (St. Petersburgh).	1875 July 27	59	M.	Carcinoma.	Entire larynx.	Death from croupous pneumonia 3 mos. after operation.	Centbl. f. Chir., 1882, No. 25, and letter from Dr. A. Schmidt to Prof. Burow (Arch. Laryng., N. Y., April, 1883).	
11	Multanowski (St. Petersburgh).	1875 Aug. 9	47	M.	Carcinoma.	Entire larynx.	Death from recurrence 2 months after operation.	As above.	
12	Billroth (Vienna).	1875 Nov. 11	54	M.	Diffuse carcinoma of the larynx.	Entire larynx.	Death on the fourth day from extensive broncho-pneumonia.	Billroth's Clinical Surgery, London, 1881, p. 133.	Had not been preceded by tracheotomy; wound did well, with but little febrile reaction.
13	Maas (Freiburg).	1876 Feb. 5	50	M.	Epithelioma of the larynx.	Entire larynx, with exception of the epiglottis and of a small piece of the cricoid cartilage.	Death from recurrence 6 months after the operation	Arch. f. klin. Chir., Bd. xx. p. 535. Private communication from operator to Dr. Foulis. Trans. Int. Med C., London, 1881.	Recurrence in posterior portion of tongue about 3 months after operat'n. Death by hemorrhage from the ulcerated mass.
14	Gerdes (Jever).	1876 Mar. 30	76	M.	Carcinoma.	Entire larynx.	Death on the fourth day from collapse.	Arch. f. klin. Chir., Bd. xxi. H. ii. p. 473.	
15	Reyher (Dorpat).	1876 May	60	M.	Carcinoma of the vocal bands.	Entire larynx, with exception of epiglottis.	Death on the eleventh day from hypostatic pneumonia.	St. Petersburgher med. Woch., 1877, Nos. 17 and 18.	

TABLE I—*continued.*

No.	Operator.	Date.	Age	Sex.	Disease.	Parts removed	Result.	Reference	Remarks.
16	Watson, P. H. (Edinburgh).	1876	60	F.	Epithelioma of larynx, with enlargement of some adjacent glands.	Larynx and enlarged glands.	Death in one week from pulmonary embolism.	Letter from operator to Dr. Foulis.	The lingual and facial veins were cut in the operation.
17	Kosinski (Warsaw).	1877 Mar. 15	36	F.	Epithelioma of the larynx, with perforation of the skin.	Entire larynx.	Death from recurrence 9 months after operation.	Centbl. f. Chir., 1877, No. xxvi. p. 401. Private communication from operator to Dr. Foulis.	
18	Foulis (Glasgow).	1877 Sept. 10	28	M.	Sarcoma, "partly papilloma, partly spindle-celled sarcoma."	Entire larynx, with exception of superior cornua of thyroid cartilage and half the arytenoid cartilages.	Death from tracheal and pulmonary phthisis, March 1, 1879.	Lancet, Oct. 13, 1877, and Mar. 29, 1879.	
19	Wegner (Berlin).	1877 Sept. 16	52	F.	Carcinoma of the larynx, size of walnut, originating from right ventricle.	Entire larynx, with epiglottis, but leaving lower half of the cricoid cartilage.	Well April 12, 1878.	Verh. der Deutsche Gesellsch. f. Chir., 1878. Private communication from operator to Dr. Foulis.	
20	Bottini (Turin).	1877 Aug. 29	48	M.	Epithelioma of the larynx.	Entire larynx and portion of œsophagus.	Death on the third day from double pneumonia.	Annales des maladies de l'oreille et du larynx, July 1, 1878; Centbl. f. Chir., 1878.	Performed "bloodlessly," with galvano-cautery blade.
21	Bruns, Victor von (Tübingen).	1878 Jan. 29	54	M.	Epithelioma of the larynx (of 5 years' duration).	Entire larynx.	Death from recurrence 9 months after operation.	Wien. med. Presse, Nov. 17, 1878. Communication from Prof. Paul Bruns to Dr. Foulis.	Preliminary tracheotomy not performed. Collapse after operation, and hyperpyrexia for a week. Voice tube employed in five weeks.

TABLE I—*continued.*

No.	Operator.	Date.	Age.	Sex.	Disease.	Parts removed.	Result.	Reference.	Remarks.
22	Rubio (Madrid).	1878 May 11	41	M.	Perichondritis of the thyroid cartilage, with necrosis.	Entire larynx.	Death on the fifth day from marasmus.	Observacion clinica, etc. Real Academia de Med., Madrid. 1878.	
23	Czerny (Heidelberg).	1878 Aug. 24	46	M.	Sarcoma in and under vocal bands, and perforating the thyroid cartilage; also involving the neighboring glands.	Entire larynx, and the diseased glands.	Death 15 months after operation (as the result of a new operation?).	Letter from operator to Dr. Foulis. Schüller, die Tracheotomie, Billroth und Lücke's Deutsche Chirurgie, 1880, p. 200.	Repeated removals of recurring masses.
24	Billroth (Vienna).	1879 Feb. 27	43	F.	Epithelioma of pharynx, larynx, and thyroid gland.	Entire larynx, with part of pharynx and œsophagus.	Death during seventh week from passage of bougie into mediastinum.	Private communication from operator to Dr. Foulis.	
25	Gussenbauer (Prague).	1879 May 24	24	M.	Carcinoma.	Entire larynx.	Death 2 months after operation from tuberculosis pulmonum.	Letter from operator to Prof. Burow, Arch. Lar., April, 1883.	
26	Macewen, Wm. (Glasgow).	1879 July 31	56	M.	Carcinoma of larynx and upper end of gullet; also a glandular mass at left side of neck.	Larynx, part of gullet, and the glandular mass.	Death in 3 days from pneumonia.	Foulis, Trans. Int. Med. Con., London, 1881.	
27	Caselli Azzio (Reggio-Emilia).	1879 Sept. 20	19	F.	Sarcoma of larynx, pharynx, palate, and base of tongue.	Entire larynx, pharynx, base of tongue, soft palate, and tonsils.	Well August, 1881 (probably still living).	Bul. del Scien. Med., Bologna, 1880, vol. v.; Centbl. f. Chir., 1880; Caselli's Reprint, Bologna, 1880. Caselli's statement at meeting Int. Med. Con., London, 1881.	Operation occupied more than 3 hours, was largely done with the galvano-caustic blade, and was attended with but little hemorrhage.

TABLE I—*continued.*

No.	Operator.	Date.	Age.	Sex	Disease.	Parts removed.	Result.	Reference.	Remarks.
28	Lange, F. (New York)	1879 Oct. 12	74	M.	Sarcoma of larynx involving the gullet.	Larynx, right cornua of hyoid bone, part of gullet.	Death from asthenia nearly seven months after operation. Recurrence.	Archives of Laryngology, N. Y., 1879, p. 56.	
29	Multanowski (St. Petersburgh).	1879 Dec. 4	60	M.	Carcinoma.	Entire larynx.	Death on fifth day from pneumonia.	Centralbl. f. Chir., 1882, No. 25. Letter from Dr. A. Schmidt to Prof. Burow, Arch. Lar., April, 1883.	
30	Langenbuch	1879	An aged female.				Death on third day from collapse.	Verh. der Deutsch Ges. f. Chir., Bd. x.	Only met by compiler in Blum's list, Arch. Gén. de méd., 1882, ii. p. 79.
31	Reyher, Carl (St. Petersburgh).	1880	48	M.	Carcinoma.	Larynx.	Death on seventh day from septic bronchopneumonia.	Wolner's Med. Jour., 1880, H. i. Letter from operator to Dr. Foulis.	
32	Thiersch (Leipsic).	1880 Feb. 26	36	M.	Carcinoma.	Entire larynx and two rings of trachea.	WELL 18 MONTHS AFTER OPERATION.	Deutsch. Ztschr. f. Chir., 1881, xvi. p. 149. Centbl. f. med. Wiss., Sept. 25, 1882. Revue mens. de Laryn., No. 82, p. 350, but accredited to Landerer.	Tracheotomy, Sept. 27, 1879. Laryngotomy for access to growths, Feb. 3, 1880.
33	Thiersch (Leipsic).	1880 April 15	52	M.	Carcinoma.	Entire larynx.	WELL 17 MONTHS AFTER OPERATION.	As above.	Tracheotomy, Mar. 2, 1880.
34	Czerny (Heidelberg).	1880 Oct. 11	47	M.	Epithelioma of larynx and superjacent soft parts.	Larynx and soft tissues in front of it.	Death from exhaustion and hemorrhage 5 months (March 25, 1881) after.	Letter from operator to Dr. Foulis.	

TABLE I—*continued.*

No.	Operator.	Date.	Age.	Sex.	Disease.	Parts removed.	Result.	Reference.	Remarks.
35	Hahn (Berlin).	1880 Oct. 23	67	M.	Carcinoma.	Larynx, all except a portion of the thyroid cartilage.	FREE FROM RECURRENCE 2 YEARS AFTER OPERATION	Letter from operator to Prof. Burow. Archives of Laryngology, April, 1883.	
36	Thiersch (Leipsic).	1880 Nov. 10	45	F.	Carcinoma of pharynx and larynx.	Larynx and part of pharynx.	Death from recurrence in 4 months (March 16, 1881).	Deutsch. Ztsch. f. Chir., 1881, xvi. p. 149.	Tracheotomy, Oct. 17, 1880. Recurrence noted within 6 weeks after operation.
37	Bircher, H. (Aarau).	1880 Dec. 3	49	F.	Scirrhus of the thyroid gland, involving the larynx.	Thyroid gland excised; 6 months later the cancer recurred, and the larynx was excised with part of the gullet.	Death in 16 days from pneumonia and gangrene of the lung.	Letter from operator to Dr. Foulis. Trans. Int. Med. Congress, 1881.	
38	Pick (London).	1881 Jan. 16	39	M.	Epithelioma of larynx (preceded by papillomata).	Larynx and epiglottis.	Death in 5 days from pleurisy and pericarditis.	Lancet, April 2, 1881, p. 541, Brit. Med. Journ., April 9, 1881, p. 562.	
39	Thiersch (Leipsic).	1881 Jan. 17	57	F.	Carcinoma of pharynx and larynx.	Entire larynx and part of pharynx.	Death on seventh day from secondary infectious pneumonia	Deutsch. Ztsch. f. Chir., 1881, Bd. xvi. p. 149.	Tracheotomy, Dec. 9, 1880.
40	Toro (Cadiz).	1881 March 9			Epithelioma of larynx.	Hyoid bone, base of tongue, and larynx.	Death on fourth day from pulmonary emphysema.	Med. Record, N. Y., August 6, 1881, p. 167.	
41	Winiwarter (Liege).	1881 April	55	F.	Carcinoma.	Entire larynx.	NO RECURRENCE 11 MONTHS AFTER OPERATION.	Clinique Chir. Univ. Liege; Monatschr. f. Ohrenheilk. 1882, No. 9. Burow's list.	
42	Foulis (Glasgow).	1881 April 30	50	M.	Epithelioma of larynx (preceded by papillomata).	Larynx.	Well and strong August, 1881.	Brit. Med. Jour., May 7, June 11, 1881; Trans. Int. Med. Congress, London, 1881.	

TABLE I—*continued.*

No.	Operator.	Date.	Age.	Sex.	Disease.	Parts removed.	Result.	Reference.	Remarks.
43	Czerny (Heidel-) berg).	1881 May 12	47	M.	Epithelioma.	Larynx and upper two rings of trachea.	Well and strong August, 1881.	Letter from operator to Dr. Foulis. Verbal report to Int. Med. Cong., London, 1881.	
44	Reyher, Carl (St. Petersburgh).	1881 May 14	57	M.	Carcinoma.	Larynx.	Death on fifth day from septic broncho-pneumonia.	Letter from operator to Dr. Foulis. Trans. Int. Med. Congress, 1881.	
45	Kocher (Berne).	1881 May 16	59	M.	Carcinoma.	Entire larynx, except a piece of the cricoid cartilage.	NO RECURRENCE 16 MONTHS AFTER OPERATION.	Letter from operator to Prof. Burow. Arch. Laryn., N.Y. April, 1883.	Patient wears a self made artificial epiglottis to overcome choking in deglutition. result of excision of epiglottis.
46	Tilanus (Amsterdam).	1881 May	51	M.	Epithelioma.	Entire larynx.	Death in 36 hours from collapse.	Centbl. f. Chir., 1882, No. 34	
47	Gussenbauer (Prague).	1881 May 19	48	M.	Carcinoma.	Entire larynx.	WELL 19 MONTHS AFTER OPERATION; doing duty as a riding-master.	Letter from operator to Prof. Burow. Arch. Laryn., N.Y. April, 1883.	
48	Volker (Brunswick).	1881 May 28	44	F.	Carcinoma epithelioides.	Entire larynx.	Death from suffocation 5 months after operation.	Academisch Proefschrift, Amsterdam, 1882, pp. 84 and 112.	Suffocated while patient had withdrawn canula to cleanse it.
49	Albert (Vienna).	1881 July 6	45	M.	Carcinoma, almost filling right half of larynx.	Entire larynx, except epiglottis; also small section of adherent œsophagus.	Death on eighth day from diffuse bronchitis and lobular pneumonia.	Wien med. Presse, 1881, xxii. p. 1373.	On 13th, hemorrhage from internal carotid, arrested by ligature above and below point of erosion. Collapse same evening; death next day.

TABLE I—*continued.*

No	Operator.	Date.	Age	Sex.	Disease.	Parts removed.	Result	Reference	Remarks.
50	Hahn (Berlin).	1881 Aug. 13	46	M.	Carcinoma.	Entire larynx.	Death in 25 days from putrid bronchitis.	Letter from operator to Prof. Burow.	
51	Margary (Turin).	1881 Sept. 29	36	F.	Epithelioma of œsophagus and larynx.	Larynx, first ring of trachea, thyroid body, part of pharynx, and œsophagus.		Arch. Ital. di Lar. Jan. 15, 1882.	Recurrence at end of 3 mos.
52	Gussenbauer (Prague).	1881 Oct.	62	M.	Carcinoma.	Entire larynx.	STRONG AND WELL 14 MONTHS AFTER OPERATION.	Letter from operator to Prof. Burow.	
53	Reyher, Carl (St. Petersburgh).	1881 Oct. 10	73	M.	Carcinoma.	Larynx and upper three rings of trachea.	Death from recurrence, 9 months after operation.	St. Petersburgh med. Ztschr., 1882, No. 28. Letter from operator to Prof. Burow.	
54	Reyher, Carl (St. Petersburgh).	1881 Oct. 10	65	M.	Carcinoma.	Larynx.	Death on seventh day from septic pneumonia.	As above.	
55	Novaro.	1881	63	M.	Carcinoma.	Entire larynx.	Recurrence in four months. Excision of right lobe of thyroid gland and part of pharynx. Death from hemorrhage on 11th day.	Giorn. di R. Acad. di med. di Torino, vol. xxxix. 1881, p. 39; Arch. Ital. di Lar., July 15, 1882.	
56	Schede.	1881	54	M.	Cancroid.	Larynx, hyoid bone, and thyroid gland.	Two months later the patient was fitted with an artificial larynx.	Deutsche med. Woch., 1882, No. 33, p. 45.	
57	Reyher, Carl (St. Petersburgh).	1882 April 7	55	M.	Carcinoma epithelioides.	Entire larynx, pharynx, and parts of œsophagus.	Death 14 days after operation from exhaustion.	Letter from operator to Prof. Burow.	
58	Kocher (Berne).	1882 May 13	54	M.	Carcinoma.	Entire larynx and carcinomatous glands.		Letter from operator to Prof. Burow.	Recurrence seven months after operation.

TABLE I—*concluded.*

No.	Operator.	Date.	Age.	Sex	Disease	Parts removed.	Result.	Reference.	Remarks.
59	Whitehead, Wm. (Manchester).	1882 May 27	46	M.	Epithelioma of right vocal band and parts subjacent (as far down as upper portion of trachea).	Thyroid and cricoid cartilages and two rings of trachea, leaving epiglottis intact.	WELL JANUARY 31, 1883. Probably still alive.	Lancet, Nov. 4, 1882, p. 741. Letter from operator to compiler, dated Jan. 31, 1883.	
60	Bergmann, von (Würzburg).	1882 June 12	54	M.	Carcinoma. (Adeno-sarcoma?).	Entire larynx.	SPEAKS WELL WITH ARTIFICIAL (BRUNS) LARYNX SOME MOS. AFTER OPERATION.	Sitzungsb. Würzburg Phys. Gesell., 1882, 47–56. Deutsche med. Woch., 1882, No. 35.; Centbl. f. Chir., Aug. 19, 1882.	
61	Burow (Königsberg).	1882 July 7	44	M.	Carcinoma.	Entire larynx without epiglottis.	Death from sudden suffocation 4½ mos. after operation.	Archives of Laryngology, April, 1883.	
62	Kocher (Berne).	1882 Sept. 28	43	M.	Carcinoma.	Entire larynx and a portion of œsophagus.		Sammlung klin. Vorträge v. Volkmann, No. 224, p. 1944.	
63	Maydl (Vienna).	1882	50	M.	Carcinoma.	Larynx, except cricoid cartilage. Excision of a gland, size of a dove's egg.	No recurrence 3 mos. after operation.	Wien med. Presse, 1882, xxiii, 1672; Wien med. Woch., 1882, No. 44. Letter from operator to Prof. Burow.	
64	Ruggi.	1882	10	M.	Polypi of the larynx.	Entire larynx.		Centbl. f. Chir., 1882, No. 45; Raccoglitore med., 1882, xviii. p. 36.	"Recovery in 28 days." Burow's list.
65	McLeod (Calcutta).	1882	35	M.	Papilloma of larynx.	Entire larynx and thyroid gland.	In good condition one month later.	Ind. Med. Gaz., 1883, xviii., 24–26.	

Of the above sixty-five complete operations, four were performed in non-malignant cases; one for cicatricial syphilitic stenosis, with death "some weeks after from pneumonia" (Case 1) [Watson]; one for necrosis, the case terminating fatally by marasmus five days after the operation (Case 22) [Rubio]; one for polypi of the larynx (Case 64) [Ruggi]; and one for papilloma of the larynx (Case 65) [McLeod].

Of the sixty-one operations remaining in this list, five were performed for sarcoma; in two of which the results were so remarkably exceptional, that attention should be especially directed to them.

I. Bottini, of Turin, on Feb. 6, 1875, removed the entire larynx from a male subject twenty-four years of age, with a laryngeal sarcoma, partly round-celled, partly spindle-celled. Notwithstanding copious hemorrhage and severe erysipelas, the patient recovered. He was reported well in August, 1881, or more than six years after the operation, and I have seen no notice of his death. He had been performing the duties of a postman, and walking eight miles a day. This is the most successful case on record.

II. Caselli, of Reggio-Emilia, on Sept. 20, 1879, removed the larynx, pharynx, base of the tongue, soft palate and tonsils, from a female subject nineteen years of age, for a sarcoma of the larynx, pharynx, palate, and base of tongue. The patient was reported well in August, 1881, practically two years after the operation, and I have seen no notice of her death. This is the second best case on record.

The remaining three patients operated upon for sarcoma died at the periods of seven, fifteen, and seventeen-and-a-half months, respectively.

TABLE II.—CASES OF SARCOMA.

1 (Lange) lived nearly 7 months.
1 (Czerny) " " 15 "
1 (Foulis) " " 17½ "
1 (Caselli) was alive Aug. 1881, nearly 2 years after operation.
1 (Bottini) was alive and well Aug. 1881, 6½ years after operation.

Taking for granted, as we are bound to do, that death was imminent in these five cases of sarcoma when the operation was resorted to, we have a considerable prolongation of life in every instance, and a remarkable prolongation in two, or in forty per cent. As far as these limited statistics go, therefore, the operation of excision of the larynx in hopeless cases of sarcoma is worthy the serious consideration of the surgeon.

The history of the remaining fifty-six operations presents a much more gloomy account. They were all for carcinoma, if we may include under that head Schede's case (56) of "cancroid."

Let me read the list of deaths as far as reported:—

TABLE III.—RECORDED DEATHS AFTER LARYNGECTOMY FOR CARCINOMA.

1.	Tilanus,	Case 46.	Death in	36	hours	from	collapse.
2.	Macewen,	" 26.	"	3	days	"	pneumonia.
3.	Bottini,	" 20.	"	3	"	"	"
4.	Langenbuch,	" 30.	"	3	"	"	collapse.
5.	Schmidt,	" 4.	"	4	"	"	"
6.	Gerdes,	" 14.	"	4	"	"	"
7.	Billroth,	" 12.	"	4	"	"	pneumonia.
8.	Toro,	" 40.	"	4	"	"	pulmonary emphysema.
9.	Schönborn,	" 7.	"	4	"	"	?
10.	Multanowski,	" 29.	"	5	"	"	pneumonia.
11.	Reyher,	" 44.	"	5	"	"	septic broncho-pneumonia.
12.	Pick,	" 38.	"	5	"	"	pleurisy and pericarditis.
13.	Reyher,	" 31.	"	7	"	"	septic broncho-pneumonia.
14.	Reyher,	" 54.	"	7	"	"	septic pneumonia.
15.	Watson,	" 16.	"	7	"	"	pulmonary embolism.
16.	Thiersch,	" 39.	"	7	"	"	"secondary infectious" pneumonia.
17.	Albert,	" 49.	"	8	"	"	"diffuse bronchitis and lobular" pneumonia.

TABLE III.—*continued.*

18. Reyher,	Case 15.	Death in 11 days	from	"hypostatic"	pneumonia.
19. Reyher,	" 57.	" 14 "	"		exhaustion.
20. Maas,	" 5.	" 2 weeks	"		pneumonia.
21. Watson,	" 6.	" 2 "	"		"
22. Bircher,	" 37.	" 16 days	"		pneumonia and "pulmonary gangrene."
23. Hahn,	" 50.	" 25 "	"		putrid bronchitis
24. Billroth,	" 24.	" 6 weeks	"		passage of bougie into mediastinum.
25. Multanowski,	" 11.	" 2 mos.	"		recurrence.
26. Gussenbauer,	" 25.	" 2 "	"		tuberculosis pulmonum.
27. Multanowski,	" 10.	" 3 "	"		"croupous" pneumonia.
28. Langenbeck,	" 9.	" 4 "	"		recurrence.
29. Thiersch,	" 36.	" 4 "	"		"
30. Novaro,	" 55.	" 4 "	"		recurrence and hemorrhage consequent upon additional operative procedures.
31. von Burow,	" 61.	" 4½ "	"		sudden suffocation.
32. Czerny,	" 34.	" 5 "	"		recurrence.
33. Volker,	" 48.	" 5 "	"		suffocation.
34. Heine,	" 3.	" 6 "	"		recurrence.
35. Maas,	" 13.	" 6 "	"		"
36. Billroth,	" 2.	" 7 "	"		"
37. Bruns,	" 21.	" 9 "	"		"
38. Kosinski,	" 17.	" 9 "	"		"
39. Reyher,	" 53.	" 9 "	"		"
40. Czerny,	" 23.	" 15 "	"		"

To this list may be added the cases of—

Margary, Case 51, in which recurrence was reported at 3 months.
Kocher, " 58, " " " 7 "

In the following cases neither death nor recurrence has been reported:—

1. Foulis, case 42, alive 5 weeks after operation.
2. Czerny, " 43, " 6 " "
3. Schede, " 56, artificial larynx adjusted 2 months after operation.
4. Mayal, " 63, no recurrence 3 months after operation.
5. von Bergmann, " 60, alive "some" " "
6. Wegner, " 19, " 7 months after operation.
7. Whitehead, " 59, "no recurrence;" alive 8 months after operation.
8. Winniwarter, " 41, " " " 11 " "
9. Gussenbauer, " 52, well 14 months after operation.
10. Kocher, " 46, no recurrence 16 months after operation.
11. Thiersch, " 33, well 17 months after operation.
12. Thiersch, " 32, " 18 " "
13. Gussenbauer, " 47, " 19 " "
14. Hahn, " 35, " 2 years " "

Of the forty reported deaths, seventeen, or forty-two-and-a-half per cent., occurred within eight days,

and five more succumbed within the second period of eight days.

The danger during the first few days is from shock and from pneumonia. Very few have perished from direct shock, very many from pneumonia. The pneumonia has been attributed generally to the ingress of blood, aliment, and septic materials into the air-passages; but, if I may form an opinion from what I have witnessed in many other operations upon the neck, there is a certain amount of risk of pneumonia in all surgical interferences in the cervical region, even when the air-passage is not opened. Thus I have seen it follow extirpation of the thyroid gland, extirpation of cervical neoplasms, and even exploratory incision into the region. It may be that the reduction of temperature to which the pneumogastric nerve is subjected leads to pneumonia, and that the manipulations within the wound render it especially sensitive. When we reflect that the majority of these operations of laryngectomy consume from one to three hours in their performance, we can fairly presume that the pneumogastric nerve is subjected to sufficient ordeal to excite an early pneumonia, quite independently of access of foreign matter to the lungs; a sequel of the operation against which every available precaution is taken.

The danger from pneumonia does not seem to exist longer than two weeks, for we have but one record of death from this cause after the sixteenth day, and that from "croupous pneumonia" (Case 10) at the end of three months. This important fortnight safely passed, the life of the patient appears comparatively secure up to the fourth month. At the fourth month

death begins to be imminent from recurrence, and we have, in our table, three at four months; one, and possibly several, within five months; two at six months; one at seven months; three at nine months; and one at fifteen months. One death from recurrence (Case 11) is reported at two months after operation. Nine cases are reported living at seven, eight, eleven, fourteen, sixteen, seventeen, eighteen, nineteen, and twenty-four months, respectively. Thus forty-two-and-one-half per cent. of the forty cases, recorded as terminating fatally, or more than thirty per cent. of the entire number operated upon for carcinoma, perished within eight days; and at the end of six months, thirty-five of the forty were dead, or eighty-seven-and-one-half per cent.: making sixty-two-and-one-half per cent. of the entire fifty-six operations for carcinoma; with great probability of a still higher percentage had all the deaths been reported.

Let us contrast this record with the average life of carcinoma of the larynx not subjected to the radical operation of laryngectomy.

Of a number of cases of carcinoma of the larynx under my own care, who agreed to submit to exsection of the larynx should I so determine, and in whom I performed tracheotomy in preference, one lived six months, two lived seven months, one lived thirteen months, and one eighteen months, respectively, after the tracheotomy.

Had laryngectomy been practised in these five cases, with equal tenure of existence, the result would have been accredited to the radical procedure. Had the operation been performed, one life might possibly have been prolonged; the majority, however, would probably have been shortened. At the period a

the end of which eighty-seven-and-a-half per cent. of the recorded deaths after laryngectomy had occurred, *i. e.*, six months, all my tracheotomized cases were living; and but two of the entire number of fifty-six excisions for carcinoma outlived the longest-lived instance in my tracheotomized list. It is not improbable that a complete series of collated statistics would present a far better exhibit for the cases merely subjected to tracheotomy and palliative procedure. It is generally believed that the natural history of carcinoma of the larynx comprises an average existence of about two years and a half; tracheotomy becoming requisite at a period varying from nine to eighteen months, according as the disease is wholly intra-laryngeal or more parietal.

In laryngectomy, the initial shock is severe, and sure to carry off a large percentage of cases by collapse, or by pneumonia. A certain number of lives are sacrificed; and the condition of the survivors, with their artificial substitutes for the larynx, is often described as pitiable in the extreme.

In tracheotomy, there is little shock, very slight danger of pneumonia, and much less risk of septic infection. Life is not likely to be sacrificed in any instance; and existence is much more comfortable after the operation than after laryngectomy.

For these reasons, excision of the larynx for carcinoma does not, in my opinion, tend to the prolongation of life; for the prolonged existence of a very few seems purchasable only at the sacrifice of the remnants of existence of many others. The greatest good to the greatest number appears better secured by dependence on the palliative operation of tracheotomy.

REPORT

OF A

CASE OF RESECTION OF THE RADIUS PERFORMED BY DR. JOHN RHEA BARTON IN 1828.

By

WM. BARTON HOPKINS, M.D.,

SURGEON TO THE OUT-DEPARTMENTS OF THE PENNSYLVANIA, EPISCOPAL, AND UNIVERSITY HOSPITALS.

[Read April 4, 1883.]

This patient came under observation a month ago at the Episcopal Hospital, where she came with her son who had received an injury of the head.

The account which she gives of an operation performed upon her fifty-four years ago, by Dr. John Rhea Barton, is in many respects incomplete; but when it is considered that more than half a century has elapsed, that she was only ten years old at the time, and that during all this interval she has never been questioned particularly in regard to the operation, or to the disease which gave rise to it, her description will appear vivid, and an adequate idea may be formed of the deep impression which was made upon her memory by undergoing a painful surgical operation without anæsthetic.

She says that when she was ten years of age she

was taken in the spring or early summer to the Pennsylvania Hospital for a disease of the forearm, which, it was thought, followed a sprain of the wrist. After remaining for some time in the house she was operated upon. Dr. Barton placed her arm upon a table, and, with an instrument which cut at both ends, made an incision, and by scraping and cutting removed a long piece of bone full of small holes like a honey-comb, and containing a large cavity in its centre. He told the students present at the operation that he did not expect new bone to form, but that a gristle-like substance would take its place. He was assisted by the Resident Physicians, Drs. James A. Washington and George Fox. She remained in the hospital until the autumn, and in a few months after her discharge regained to a great extent the use of her hand.

Her name and age have been found in the record of the hospital; the date of her admission being July

16, 1828, and of her discharge, November 15 of the same year.

In studying the condition of the parts as they now present themselves it will be of interest to note the main causes which have brought about the usefulness of the hand, the changes in position and function of the parts not involved in former disease incident to the removal of so important a structure as the entire or almost entire diaphysis of the radius, and the influence these alterations have had upon the growth and development of the bones and soft parts. The usefulness of the hand would seem to depend not only upon the preservation of the upper and lower epiphyses of the radius, the former through probable extension of the periosteum giving attachment to the biceps muscle and the latter to the long supinator, but also upon the strong healthy character assumed by the skin investing the lower end of the ulna. This skin-cap is the principal antagonist to the action of the muscles of the forearm, and if from constant pressure against the bone it had become thin and tender, as it very likely would have become without the intervention of a bursal sac, every movement of these muscles would have caused pain, and the hand would have become almost useless.

The skin has, of course, yielded to a considerable extent, and the hand has, therefore, been gradually dragged up the forearm. The thumb, from the loss of the origin of its long flexor, has become feeble and disused. The ulna has been made an active agent in support, and having lost its styloid process, presumably by absorption, the lower end has assumed a smooth, knob-like appearance.

The growth of the sound bones has been comparatively little arrested, this being evidence that active work was performed by the hand during the growing years which succeeded the operation. The humeri are of the same length. The right ulna is one-and-a-quarter inches shorter than the left. Part of this difference must be accounted for, however, by the absence of the styloid process, and part may also be due to a somewhat deficient development of the whole lower epiphysis in length—though not in breadth—as it is full and broad.

From measurements made of the ulna in connection with this case, the average length of the bone in females at the age of ten years is about seven-and-a-half inches. In this case it is ten-and-a-quarter inches upon the left side, and nine upon the right. The difference in the circumference in the middle of the arms is three-quarters of an inch; in the middle of the forearms, two inches; and of the hands, one inch; all in favor of the left or sound side.

The hand is set in an everted position well up the forearm, the tip of the middle finger being four inches and a quarter nearer the elbow than upon the left side. Although thus distorted, and so loosely attached that it can be brought at right angles with the line of the ulna, it is remarkably strong, and with it she is capable of executing almost any movement.

Articulated with the carpus is felt the lower end of the radius. In its proper place the head of the radius may also be felt, continuous with which, and extending down the forearm nearly to the hand, is a prolongation of fibro-cartilage, occupying the site of the excised bone.

Exactly where this new formation begins, and hence, how much bone was removed by the operation, it is difficult to determine; but in regard to this, as well as the other points of interest which have been alluded to, you are invited to examine for yourselves in the patient whom I now present.

INFANT FOODS.

By

Prof. ALBERT R. LEEDS, Ph.D.,

of the Stevens Institute of Technology, Hoboken, N. J.

[Read, by invitation, May 2, 1883.]

In attempting to discuss the problem of Infant Foods, I shall begin by assuming that the natural food of the human infant is the best infant food. Without pausing to give the grounds on which it is based, I shall merely add, that the conviction has constantly strengthened, with the progress of chemical analyses of the human milk and of the various substitutes which have been proposed, that the above assumption is the safest and least presumptuous one which can be made in the present stage of theoretic and practical knowledge.

But what is human milk? To answer this question satisfactorily we should know at least three things: 1st. All the components; 2d. Their relative proportion; 3d. Their chemical and physiological properties.

Strangely enough, present knowledge on all these points is very far from satisfactory. With reference to the first point, it is sufficient to state that no complete analysis of woman's milk has yet been made.

As to the second, I am not referring to the fact that the milk necessarily varies with the interval since parturition, the nutrition and constitution of the mother, and numerous other variable elements, but to the much more important one, that the average composition of the milk of healthy nursing women is by no means satisfactorily established. You will be surprised, I think, on turning to the literature of this subject, to find how widely different are the figures obtained by investigators, and variously adopted, usually without critical consideration, in works treating of the nutrition and diseases of children. Finally, in reference to the third point, our knowledge of the properties of the components themselves is extremely meagre. We know, as yet, very little concerning the true nature of the nitrogenous bodies, which are lumped together under the head of albuminoids. And as to the fat of human milk, no separation into the many oily and fatty bodies, of which it is made up, has as yet been attempted.

It may be said that this is foolish hypercriticism—that if we do not know everything, we know sufficient to conduct the nutrition of infants on a sound basis of adequate knowledge. But many reasons could be given under the heads previously specified for regarding the last assumption premature. Our time will not permit us even to allude to these, but will enable us to speak only of the one practical issue which it is our business to deal with this evening. This is, granting that woman's milk is the best infants' food, in what manner should the nature and proportions of the components be determined of any substitute we may be necessitated to employ? Certainly, only by

knowing, in the first place, the average composition of human milk.

As this point is fundamental, and as the wide diversities I have just alluded to existed concerning it, I have thought it well to devote the past four months to its study. In this labor I have had the cordial co-operation of Dr. A. M. Thomas, Chief of Medical Staff of the Emigrants' Asylum and Hospital, and, during the later part of the inquiry, of Dr. K. Parker, of the Infants' Asylum of New York. Both of them have given their personal attention to the collection of the samples, which, in every case, were taken from healthy women, mainly young and mostly primiparae. All the points of interest, with regard to the history of the woman, the child, and the sample, have been tabulated and co-ordinated, and will probably be published in connection with a future monograph devoted to the topic of human milk. This would not be a proper place for these details, further than adding that the samples usually amounted to two ounces, were taken in most instances two hours after time of last nursing, and were generally the entire contents of the gland.

The sources of variation in the multitudinous analyses of woman's milk that have been previously published, are principally three in number: the most important being the differences in the methods of analyses; next, the variations in the constitution of the milk itself; lastly, the circumstances connected with the collection of the sample. Without delaying to discuss these matters, which would require much time and precision of statement, I shall bring before you to-night only so much of the results thus far ob-

tained as are needed for our present purpose. They are to be regarded as an approximation, to be still further modified by incorporation of the results of other analyses still in progress.

ANALYSES OF 43 SAMPLES OF WOMAN'S MILK.

Reaction uniformly alkaline.

	Average.	Minimum.	Maximum.
Specific gravity	1.0317	1.030	1.0353
Water	86.766	83.34	89.09
Total solids	13.234	10.91	16.66
Total solids not fat	9.221	6.57	12.09
Fat	4.013	2.11	6.89
Milk-sugar	6.997	5.40	7.92
Albuminoids	2.058	0.85	4.86
Ash	0.21	0.13	0.35

Not only was the reaction alkaline when the samples were fresh, but with one exception in which it was neutral, this alkalinity was found to remain twenty-four or more hours after.

The most striking feature in these analyses is the great range of variation in the amounts of certain constituents, more especially in the albuminoids, the maximum, 4.86 per cent., being nearly six times the minimum, which is only 0.85 per cent. The next most variable constituent is the fat, the maximum being more than three times the minimum. Then come the saline matters, nearly three, and last of all the milk-sugar, which differs but little from the mean (6.997 per cent.) in most samples. In other words, the most striking peculiarity in woman's milk is not the constancy but the great variability in its composition. Furthermore, this variability is by far the greatest in the constituent most essential to nutrition, the albuminoids, and diminishes in degree of variability in proportion as the constituent becomes less and less essential to nutrition, becoming very nearly constant

in the case of milk-sugar, the function of which is not nutrition but heating. By which nature appears to intend to teach us that the rate of nutrition in an infant may safely vary within wide limits, but that the animal heat of a highly organized and rapidly developing creature must be maintained *coûte qu'il coûte*. To maintain at a temperature somewhat exceeding that of an adult, a creature whose vital processes on the one hand are of great activity, while on the other hand the supplies of heat due to cerebral impulses and self-originated locomotions are extremely small, requires the rapid consumption of available fuel, and the abundance of carbohydrates in the milk supplies this most imperative want.

Without stopping to draw out the significance contained in the proportionality of the constituents, both as considered in themselves and as compared with the new-born of other mammalia, let us hasten on to the comparison of human milk with its nearest analogue. This is properly asses' milk, but as this is not and never will be generally available, it will be more profitable for us to consider the cheap and universally accessible cow's milk. For a similar reason, I shall not institute a comparison between the above average and that of the rich milk of blooded cattle, the Alderneys, Jerseys, and so on, nor with selected samples from the best ordinary milch-cows. Rather, as such comparison will be of greater practical utility, I shall adduce the analyses of unadulterated whole commercial, or "market" milk. And as this market milk is itself the average of a great number of samples, it is useless, so far as it is concerned, to give the maxima and minima of its individual constituents.

As determined by methods identical with those employed in the analyses of woman's milk, I shall state, therefore, the following results, obtained upon samples of unadulterated cow's milk, such as is sold by farmers in New Jersey to the citizens of New York and Philadelphia.

ANALYSES OF 11 SAMPLES OF WHOLE MARKET MILK.

Water	87.7	per cent.
Total solids	12.3	"
Total solids not fat	8.48	"
Fat	3.75	"
Milk-sugar	4.42	"
Albuminoids	3.42	"
Ash	0.64	"

As in the case of woman's milk, the slight discrepancies noticeable are due to the fact that the figures for "total solids" were those obtained by appropriate separate determinations, and of course do not precisely agree with the figures obtained by mere addition of the solid constituents.

Before proceeding to make a comparison, I wish to quote here the results given by König (*Chemie der mensch. Nahrungs- und Genussmittel*), deduced in each case from the analyses of many hundred samples of woman's and cow's milk, and to call attention to the fact that whilst these results include determinations effected by very many and diverse methods of all descriptions of samples, and present a distinction between caseine and albumen (a distinction which cannot be sharply effected by present analytical methods), yet as a whole they are similar to and support the inductions which I shall base upon the analyses above given.

WOMAN'S MILK.

	Mean.	Minimum.	Maximum.
Water	87.09	83.69	90.90
Total solids	12.91	9.10	16.31
Fat	3.90	1.71	7.60
Milk-sugar	6.04	4.11	7.80
Caseine	0.63	0.18	1.90
Albumen	1.31	0.39	2.35
Albuminoids	1.94	0.57	4.25
Ash	0.49	0.14	?

COW'S MILK.

	Mean.	Minimum.	Maximum.
Water	87.41	80.32	91.50
Total solids	12.59	8.50	19.68
Fat	3.66	1.15	7.09
Milk-sugar	4.92	3.20	5.67
Caseine	3.01	1.17	7.40
Albumen	0.75	0.21	5.04
Albuminoids	3.76	1.38	12.44
Ash	0.70	0.50	0.87

The same striking peculiarities are noticeable in the above analyses of woman's milk. The greatest variable is the albuminoid constituent, the maximum being more than seven times the minimum; the most nearly constant is the milk-sugar, varying little from the mean of 6.04 per cent., which is likewise the largest of the solid constituents.

When we compare woman's with cow's milk, it is the great differences and not the similarities which surprise us, and demand study, recognition, and utilization in the solution of the problem of artificial infant's food. In woman's milk we have a persistently alkaline liquid, of a somewhat animal, usually disagreeable, and very rarely sweetish taste, of somewhat greater specific gravity (1.0317) than cow's milk (1.029). Although it has less water, and greater total solids, and total solids not fat, than cow's milk, it is by no means so opaque, and with its thin and watery consistence gives us a notion the reverse of true with

regard to its real composition. Agreeing with cow's milk in the fact that the milk-sugar in both is the chief solid, it differs in that its milk-sugar largely exceeds the milk-sugar of cow's milk. It likewise exceeds in fat. In albuminoids it falls far below. And whilst by present modes of analysis the separation of the so-called caseine from the so-called albumen is not accurately performed, yet the results are approximately correct, and have a very great value in pointing out the most important of all the differences between the two secretions, which is that the fraction of the total albuminoids in cow's milk which is coagulable by acids is far greater (perhaps four times) than the non-coagulable part.

In woman's milk, on the contrary, the reverse is true, and the non-coagulable part much exceeds (perhaps by more than twice) the coagulable portion. And whilst the absolute amount of ash is less, the relative amount of potash is greater, in woman's than in cow's milk.

For reasons which will appear further on, it would seem that the best solution of the problem of artificial infant feeding is to be found in the substitution of cow's for human milk. But, inasmuch as the secretion of the herbivora is radically and in all particulars different from that of the omnivora, cow's milk is profoundly altered so as to assimilate, in the ratio and nature of its constituents, human milk.

To discuss the various methods by which it has been proposed to effect this result would far outrun my present scope, which is only so much of the general discussion as relates to the influence exerted upon cow's milk by the addition of the various articles of

infant food now manufactured and offered for sale. This influence, we shall find, is chiefly of a mechanical or physical character, and does not necessarily involve the discussion of those chemical changes, at present time under investigation in many quarters, the practical results of which have not yet been satisfactorily determined, and belong, therefore, to the future of this subject.

The method hitherto employed is spoken of as mechanical for reasons based partly on the practice of physicians, and partly on laboratory experiments. The mere addition of water to cow's milk is sufficient to reduce the percentage of albuminoids to the same amount as its percentage in human milk. But this addition does little to diminish the size and compact character of the clot of cow's milk. This last is effected, as far as it actually is effected, which is only partially, by the addition of the various attenuants composing manufactured infant's food, whether that attenuant is starch, gum, sugar, dextrine, or other bland nutrient. This explanation of the utility of manufactured infant's foods accounts for the seeming anomalies in present medical practice, which at first sight appear very startling and inconsistent with generally accepted physiological doctrines. For whilst admitting that the secretions of the salivary and pancreatic glands are insufficient in the early stages of infancy to convert more than very limited amounts of starch, yet physicians frequently use with good results a farinaceous food like Ridge's, which contains 77.96 per cent. of starch, or like Robinson's patent barley, which contains 77.76 per cent. of starch. But when we consider that the utility of

this starch is not in the way of infant's food, for which it is not adapted, but as an attenuant of the large amount of diluted milk with which it is mixed, then the seeming contradiction between theory and practice disappears.

To discover whether this interpretation is in accord with experiment, the coagulation was effected in presence of similar attenuants. In the first place, the total albuminoids were determined in a sample of whole cow's milk, and were found to be 3.39 per cent. The so-called caseine was then separated by coagulation with acetic acid, and amounted to 2.42 per cent. On boiling the filtrate, 0.26 per cent. of albumen so called separated out, leaving a deficiency of 0.71 per cent. of albuminoids to be accounted for. A direct determination of the albuminoids in the filtrate from the albumen, gave an additional yield of 0.76 per cent., showing that both coagulation and boiling of the filtrate subsequently, had left nearly one-fourth of the total albuminoids in solution.

Ten grams of the same milk, together with 25 grams cane-sugar and 110 c. c. of water, were treated in like manner, the precipitates being exhaustively washed. I obtained:

As precipitated by acid	3.13 per cent.
" " by boiling	0.40 "
Precipitated by copper sulphate from filtrate .	1.14 "
	4.67 "
Total albuminoids	3.39 "
	1.28 " .

In other words, the precipitates carried down with them 1.28 per cent. of saccharine matter, which could not be removed by washing.

Some barley-water was then made and filtered through ordinary Swedish filter paper, the clear filtrate being used in the following experiment. 10 grams of the same milk as before were mixed with 110 c. c. of the barley-water. I obtained:

As precipitated by acid	5.21	per cent.
" " by boiling	0.37	"
" " by copper sulphate from filtrate	1.35	"
	6.93	"

That is to say, the precipitates carried down with them from the clear barley-water 3.54 per cent. of barley extract which could not be removed by washing. In this case, the attenuants of the clot exceeded in weight the coagula themselves.

An experiment with grape-sugar yielded results closely resembling those with cane. With gelatine a very remarkable result was obtained. 10 grams of the same milk were added to 110 c. c. of a solution of 1 part of gelatine in 150 parts of water. Although the gelatine was so attenuated, it entirely prevented the precipitation of caseine on the addition of acid, and what is likewise interesting, appeared to arrest decomposition, the white jelly not having altered after a week's standing.

Ten grams of the same milk were added to 110 c. c. of clear starch-water (filtered through Swedish filter paper). I obtained:

As precipitated by acid	3.07	per cent.
" " by boiling	0.36	"
" " by copper sulphate in the filtrate	1.08	"
	4.57	"

Or 1.12 per cent. of starch carried down and not separable by exhaustive washing.

The utility of diluting cow's milk until its percentage of albuminoids does not exceed that of human milk, and adding some bland attenuant, is obvious. But the special virtues of the *extractive* of barley or oat-meal, as compared with starch, and the relative values as nutrients of sugar, gum, dextrine, gelatine, barley, oat-meal, etc. etc., and their relative advantages when thus employed, have been very imperfectly determined. It is much to be desired that new physiological and chemical experiments directed especially to these all-important factors in infant nutrition should be instituted. I shall have occasion to refer to the same points in connection with Liebig's Foods.

An examination of the great variety of infant foods, the analyses of which are given in the following pages, shows that they can be classified most conveniently under the three heads of Farinaceous, Liebig's Foods, and Milk Foods (including condensed and preserved milk).

I. *Farinaceous.*

A. Wheat, previously prepared by baking, including:

1. Blair's Prepared Wheat Food; 2. Hubbell's Prepared Wheat Food; 3. Imperial Granum; 4. Ridge's Food.

B. Mixtures of various cereals.

5. "A B C." Cereal Cream; 6. "A B C." Cereal Milk; 7. Robinson's Patent Barley.

II. *Liebig's Foods.*

8. Mellin's; 9. Hawleys; 10. Horlick's; 11. Keasbey and Mattison's; 12. Savory and Moore's; 13. Baby Sup No. 1; 14. Baby Sup No. 2.

III. *Milk Foods.*

15. Nestlé's; 16. Anglo-Swiss; 17. Gerber's; 18. American Swiss.

CLASS I.—A.

	1. Blair's wheat food.	2. Hubbell's wheat food.	3. Imperial granum.	4. Ridge's food.
Water	9.85	7.78	5.49	9.23
Fat	1.56	0.41	1.01	0.63
Grape-sugar	1.75	7.56	trace	2.40
Cane-sugar	1.71	4.87	trace	2.20
Starch	64.80	67.60	78.93	77.96
Soluble carbohydrates	13.69	14.29	3.56	5.19
Albuminoids	7.16	10.13	10.51	9.24
Gum, cellulose, etc.	2.94	undeterm'd	0.50	
Ash	1.06	1.00	1.16	0.60

1. *Blair's Wheat Food.*—It is claimed that this is prepared from choice wheat in such a manner as to retain all the nutritive constituents and reject those which are irritating or otherwise objectionable. Moreover, that by cooking such physical and chemical changes have been brought about as to facilitate mastication and the subsequent action of the fluids of the stomach, thereby rendering the food more easily digested. It is stated to be especially beneficial in intestinal diseases like dysentery, cholera infantum, etc.

Uncooked, this flour has a sweet pleasant taste. When cooked according to directions it forms a very smooth paste with a faint tinge of color, resembling arrowroot in its flavor and quite palatable without the addition of salt, sugar, milk, or other accompaniments.

2. *Hubbell's Wheat Flour.*—Claimed to be made from wheat alone, floured and carefully baked from eight to ten hours, at about the temperature of boiling

water. "It includes all the flesh-forming constituents, earthy and saline elements of the grain, with only a portion of the starch, and none of the silicated coating. It keeps without change."

This flour is quite sweet and palatable even in its uncooked form, and when moistened with the saliva is more pasty than the Blair's wheat flour. When cooked it forms a perfectly white smooth paste, with a very delicate flavor. It is more starch-like in consistency than Blair's, a difference due in part to the larger percentage of starch, and less pronounced in flavor, this being probably due in some degree to the smaller percentage of fat. In both Blair's and Hubbell's the per cent. of gum, cellulose, etc., is extremely small, in the latter so small that it was not determined. In nitrogen Hubbell's is much richer than either of the other two preparations, and its value for purposes of nutrition correspondingly greater. The reaction of Blair's food and of Hubbell's is in each neutral.

The excess in the amount of saccharine matter in Hubbell's food above that contained in ordinary wheat flour induced me to write for particulars of the change which it had undergone. The process I was informed is as follows: A large baker's oven is heated to about 340° to 360° F. The flour contained in shallow Russia iron pans is then put in, the fire having meantime been withdrawn, the oven closed and the flour left there about twenty-four hours. When the oven is reopened the temperature will have fallen to 100°, and after sieving, the prepared flour will be ready for use. The flour used is the best grade as made by the roller process, the second grade containing more starch, less gluten, being that bought and used by bakers.

The two following analyses, the first of Hubbell's prepared wheat flour, the second of the flour from which the first was obtained by the process of baking as conducted in the manner above described, are interesting and important as elucidating the nature of the changes thus induced.

	Wheat flour.	Same baked.
Water	9.02	7.78
Fat	1.01	0.11
Grape-sugar	2.34	7.56
Cane-sugar	2.46	4.87[1]
Starch	76.07	67.60
Soluble carbohydrates	5.66	14.29
Albuminoids	7.47	10.13

It will be seen that the flour has lost moisture in baking, and also a portion of its fat. These changes, however, are of little moment compared with the considerable decrease of starch and its conversion into saccharine bodies. The soluble carbohydrates are considerably more than doubled, and this change is one of the greatest value and importance, so far as the dietetic value of the prepared food is concerned. The considerable increase in the percentage of albuminoids I am unable to account for.

3. *Imperial Granum.*—It is stated to be "in composition principally the gluten derived by chemical process from very superior growths of wheat—a solid extract." Dr. Fowler states (*Amer. Jour. Obstetrics*, April, 1882), as the result of his microscopical examination, that if the material from which this preparation is derived contains any gluten at all, the "chemical process" resorted to in order to extract it has at the same time either destroyed it, or so altered its character as to render it no longer recognizable by the usual tests. This is an excellent illustration of

[1] With some dextrine.

the difficulty which is encountered in deciding with the microscope upon the constitution of a cereal after treatment, for whilst Dr. Fowler's statement of the microscopic appearance is in accordance with my own observations, yet as a matter of fact the Imperial Granum contains 10.51 per cent. of albuminoids. On the other hand this is not sufficient by any means to bear out the statement that the Imperial Granum consists principally of gluten. According to Dr. Fowler, it is simply coarse barley flour. My own observations make it a wheaten preparation.

4. *Ridge's Food.*—It is advertised as "prepared from carefully selected winter wheat," reduced to an almost uniform fineness. The product is then thoroughly cooked by a steam-baking process, which gradually changes a large proportion of the starch into dextrine, excluding only the woody fibre. It is afterwards rendered a little sweet and slightly alkaline.

Dr. Fowler states (*loc. cit.*) that the Ridge's food is apparently barley flour finely ground, and that the odor, dough, and microscopic appearance indicate no other ingredients. I have placed it, in accordance with my own observations, among the wheat preparations. Both Imperial Granum and Ridge's food when cooked are very palatable. Both have a neutral reaction. Both have a considerable percentage of albuminoids, that of Imperial Granum in the two samples analyzed being the higher, and both have a very high percentage of starch.

It should be very carefully borne in mind that wheat flour after carefully baking is extensively altered, and that the albuminous bodies become considerably more soluble in water. A wheat flour, which in its original

condition would yield after baking a very considerable amount of crude gluten on washing, after baking will leave a much smaller quantity, and for this reason the percentage of crude gluten in baked flours cannot be roughly estimated by washing and drying. For the same reason a baked wheat flour may be mistaken for barley flour, which gives a non-glutinous dough.

CLASS I.—B. MIXTURE OF VARIOUS CEREALS.

	6. "A B C." Cereal milk.	7. Robinson's Patent barley.
Moisture	9.33	10.10
Fat	1.01	0.97
Grape-sugar	4.60	3.08
Cane-sugar	15.40	0.90
Starch	58.42	77.76
Soluble carbohydrates	20.00	4 11
Albuminoids	11.08	5.13
Cellulose, gum, etc.	1.16	1.93
Ash	.....	1.93

5. *"A B C." Cereal Cream.*—Stated to be "prepared from the most nutritious and digestible parts of the choicest wheat and barley, with all impurities removed." It appears to be a coarse meal of wheat and barley, but I did not analyze it, the box which I purchased being probably old and its contents spoiled.

6. *"A B C." Cereal Milk.*—"Prepared by a scientific admixture of the nitrates and phosphates of wheat with the whole barley; and after adding the required sugar, we have secured an analysis almost identical with human milk. The wheat is first cleansed, then hulled, coarsely ground, and the surplus starch removed, leaving the nitrates and phosphates. The barley is hulled, crushed, and mixed with a proper proportion of the wheat nitrates and phosphates. The mixture is cooked by steam, desiccated, ground into fine flour, specks bolted out, and the requisite amount of sugar added."

The statement that this food corresponds nearly with human milk in its nutritive ingredients is untrue. The proportion between its various constituents is entirely unlike that in human milk, and more than half consists of starch, a body entirely foreign to milk.

7. *Robinson's Patent Barley*—"Patent barley, technically, is ground pearl barley. Yet this preparation, while possessing most of the characters of what it purports to be, is somewhat unlike pure barley flour. Its dough is more adhesive, and the white color, together with the mild barley odor, suggest the admixture of wheat flour. No gluten cells are seen, but there are numerous granules unaffected by iodine, and turned red by carmine (albuminous matter). The microscopic examination shows starch granules free and in bundles, held together by the cellulose. The larger corpuscles are probably those of wheat." I have adopted this description of Dr. Fowler, although I am inclined from my own observations to regard the preparation as merely barley flour.

CLASS II. LIEBIG'S FOODS.

	8. Mellin's.	9. Hawley's.	10. Horlick's.	11 Keasbey and Mattison's.	12. Savory and Moore's.	13. Baby Sup No. 1.	14. Baby sup No. 2.
Water	5.09	6.60	3.39	27.95	8.34	5.54	11.48
Fat	0.15	0.61	0.08	None	0.40	1.28	0.62
Grape-sugar	44.69	40.55	34.99	36.75	20.41	2.20	2.44
Cane-sugar[1]	3.51	3.44	12.45	7.58	9.08	11.70	2.48
Starch	None	10.97	None	None	36.36	61.99	51.95
Soluble carbohydrates	85.44	76.54	87.20	71.50	44.83	14.35	22.79
Albuminoids	5.95	5.38	6.71	None	9.63	9.75	7.92
Cellulose, gum, etc.	.....	.....	.....	.....	0.44	7.09	5.24
Ash	1.89	1.50	1.28	0.93	0.89	Undet.	1.59

[1] These determinations are probably higher than they should be, owing to the influence of dextrine upon the sugar determinations, but if there is an error it is of the same amount in every case.

The first three are dried foods, in brown or granular masses, and very sweet; the Mellin's food looking and tasting very much like pulverized candy. Their aqueous solutions, besides this sweet, had an after taste of alkaline salt. Under the microscope Horlick's food exhibited very few starch granules, some cellulose, hairs of wheat, but mostly dark bundles of entirely unrecognizable granular matters, probably converted starch. Mellin's food goes almost entirely into solution, and I failed to recognize under the microscope the minute irregular granular matter left behind. The materials sent to me by the manufacturers of Horlick's food, as representing their regular consumption, consisted of fine white wheat flour not baked, good barley malt, and pure bicarbonates of soda and potash. Singularly enough, the reaction of the Horlick food analyzed was acid; that of the Hawley's food was acid likewise, while the Mellin's food was alkaline.

The analyses reveal certain striking points in connection with these Liebig's foods. The percentage of fat is extremely low, that of grape-sugar very high. In Horlick's and Mellin's there is no unconverted starch, in Hawley's 11 per cent. In Mellin's there were 5.95 per cent. albuminoids, and, in both the others, the percentage was very low; that in Horlick's, the larger, being but 6.71 per cent. Hawley's is very light colored, less sweet than the other two, is more finely powdered, and has a smell resembling that of a cereal which has been submitted to torrefaction. The 10.97 per cent. of starch indicates either that the wheat and barley malt have undergone very partial conversion into grape-sugar, or that this percent-

age of starch has been added in some form after the liquid products of the conversion have been evaporated to dryness.

In my first paper upon this subject (*Sixth Annual Report of the New Jersey State Board of Health*) I fell inadvertently into an error in speaking of Mellin's food. The original table (p. 206 of that article) stated that this food contained 5.95 per cent. of albuminoids, which was correct. Subsequently, in writing out the report, a second table was prepared for convenience in writing out my work, and into this table the clerical blunder was introduced of including the albuminoids, 5.95 per cent., along with the insoluble residue, 3.46 per cent., and putting down the sum, 9.41 per cent. under the latter head, and thus leaving zero for the percentage of albuminoids. This error I am very glad to correct in this place, as well as the misapprehension into which it led me, that the supposed absence of albuminoids indicated the failure to use wheat and barley malt in the preparation of this food. On the contrary, the constitution of the food shows that both these articles were used, together with bicarbonate of potash, and the Mellin's is a genuine Liebig's food.

In fact, the percentage of albuminoids in a Liebig's food has not the same relative significance as it has in the case of Class I., or Farinaceous Foods. This point may be best explained by an examination of the analyses of Savory & Moore's Infant Food and the Baby Sups. These are composed of the materials out of which, by a long and tedious process of conversion, a food like Nos. 8, 9, and 10 is made. This process is as follows: Equal parts of wheat flour and barley malt, together with a certain amount of wheat bran,

added, it is said, for the sake of the adherent phosphates and nitrogenous matter, together with one per cent. of bicarbonate of potash, are mixed with sufficient water to make a thin paste. The mixture is allowed to stand at ordinary temperatures for several hours, and then heated to 150 until the conversion of the starch is completed. It is then strained, and the residue pressed and exhausted with warm water. The extract is evaporated *in vacuo* at as low a temperature as consistent with rapidity of working, dried at a higher temperature, and finally, by very skilful manipulation, brought to a finely pulverulent mass, which does not contain sufficient moisture to cake together, and is a most commendable product of technical skill.

By careful selection of highly albuminous wheat and excellent barley the best results possible can be obtained. What they are, and whether or no the addition of bran increases the percentages of phosphates and other valuable constituents in the extract, I am not prepared to say. I should have very gladly complied with the wishes of manufacturers of Liebig's foods to investigate these matters for them as a matter of ordinary professional labor, but felt that my candid expressions with regard to this subject would not permit me to do so without laying myself open to the imputation of bias. But these explanations suffice to show that the percentages of albuminoids in Liebig's foods have not the same significance as in other classes of infants' foods. The starch has undergone conversion into dextrine and grape-sugar, which, being soluble, pass into the liquid extract, and are evaporated down and pass into the final product.

But only that portion of the albuminoids which is contained in particles so fine as to pass through the strainers, together with the soluble albuminous matter (and this is especially valuable in the nutrition of infants), is found in the final product. This deficiency in total albuminoids therefore is inevitable, and is an evidence of the genuineness of a Liebig's food. To determine how large a proportion of these albuminoids passes into *solution* would be of great value. To this end a mode of analysis should be used for these foods, specially designed for them, and this analysis should give not only the varieties of sugar, the dextrine, and soluble and insoluble albuminoids, but the peculiar kinds of extractive. If the virtue of Liebig's foods resides only in their not containing starch, but its products of transformation, gum, and dextrine, together with sufficient bicarbonate of potash to make the reaction of the infant food, of milk, always alkaline, then so tedious and expensive a process would not have to be resorted to in order to obtain a mixture which can be made directly very cheaply. If, on the other hand, the other substances extracted from the grain have an important dietetic value of their own, then the analysis should be directed to ascertain their nature and amount. This has hitherto not been done, nor do we possess the records of physiological experiments bearing expressly upon these points of inquiry. In Savory and Moore's food this process of conversion has not been effected. Neither can it be when the food is prepared according to directions. These are to put one or two tablespoonfuls of the food into a suitable vessel, mix gradually with two or three tablespoonfuls of boiling water or milk, and cool to

the temperature of new milk. The conversion of the 36.36 per cent. of starch cannot be effected so readily. If it be (and it is stated that there is no objection (!) to so doing) heated until it thickens, afterwards removed from the fire and stirred until it become fluid, then a greater amount of starch will be converted than when mixed with boiling water only, but there is no certainty nor probability that, in any case, all the starch will undergo conversion. Neither its composition nor its method of preparation explain, why, when this food is prepared with water, the food is equivalent to mother's food in nourishing power, and why, as the advertisement likewise claims, when prepared with milk only, it has twice the strength. It is certainly not to the 36.36 per cent. of starch that it owes this power, nor to the 9.63 per cent. of the albuminoids of wheat and barley. In fact, this large proportion of albuminoids shows how much less of wheat and barley has actually been employed in the preparation of Savory and Moore's food, than should have been employed in order to obtain a genuine Liebig's food. For, in the latter, the fluid extract of the wheat and barley is used, and the residual matter, which is not beneficial but injurious in the nutrition of children, is strained off, so that the solid matter remaining after evaporation represents an amount of wheat and barley originally taken greatly in excess of its own weight. In Savory and Moore's food, the percentage of albuminoids is that corresponding to the sum of the weights of the ground barley malt and wheat originally taken. These are added together, along with the alkaline bicarbonate, and, when prepared for use, the insoluble albuminoids and the useless and

irritating particles, which Liebig directed should be strained off, are allowed to remain in the food.

I have spoken thus severely of Savory and Moore's food, because, whilst claiming to be prepared in accordance with Liebig's principles, it actually contravenes them. The following much less ambitious preparations are worthy of more favorable comment :—

Baby Sup No. 1 is advertised as an excellent substitute for mother's milk, in case of infants under four months of age. It is a very sweet partly crushed whole oat-meal, very palatable even before cooking, and dissolving readily in the juices of the mouth. It is prepared from malted oats, and, after the conversion of the starch has gone as far as it is thought it will proceed, the oats are carefully hulled, only a residue of the coat being left in the crack of the grain. The analysis shows the lowered percentage of starch, and the increase of saccharine bodies due to this treatment.

Baby Sup No. 2 consists of wheat flour, malted barley and bicarbonate of potash, in the proportions given in Liebig's formula. In its dry state the mixture has little taste, but becomes thin, sweet, and palatable on cooking. The analysis gives but a partial result of this change, because the food was cooked only five minutes before the analysis, whilst the directions call for a half hour's cooking. But already much of the starch has been converted into dextrine. These foods are commendable efforts to carry Liebig's views into practice, and it is to be regretted that a certain amount of care and time is required to properly cook them, and, for this reason, they will prob-

ably have only a restricted use. In Mellin's, Horlick's, and Hawley's foods this care and time have already been expended by the manufacturer, and experience has shown that few mothers or nurses can or will take so much trouble.

Keasbey and Mattison's Food.—From the point of view above taken, it is possible to explain a peculiarity in its composition, which I failed at first to understand. This is, that the sample of this food which I examined contained no albuminoids whatsoever. Its advertisement states that it is an extract prepared from malted grain, dextrine, alkaline phosphates, etc., and that it is perfectly free from starch. It does not resemble the other preparations of Liebig's foods in the market, being, unlike them, a thick liquid, resembling, both in taste and appearance, some variety of molasses or syrup. My first explanation of the entire absence of albuminoids was, that no grain was used in its preparation. This is not the case. The food is not strictly a Liebig's food, but in reality a neutralized extract of malted barley. The ground malt is extracted with hot water, the liquid strained, evaporated *in vacuo*, and neutralized with bicarbonate of potash. There is no question that the albuminoids did not pass the sieve, for I could neither determine them by ordinary methods, nor get the reaction for nitrogen by the most severe tests. But, as before stated, there must be present certain extractives, and the analysis should be directed to finding them, and thus seek to explain the undoubted benefits frequently conferred by the use of this food.

Before concluding with regard to Liebig's foods, I desire to say a few words concerning the percentages

of dextrine and cane-sugar reported for these foods. The former would be nearly that which would correspond to the difference between the amount of soluble carbohydrates and the sum total of grape-sugar and cane-sugar. But it is not put down in the analysis, because it was not directly determined, and I did not know how much soluble matter of other kinds, possibly soluble albuminoids and other extractive matter of great dietetic value, might possibly be thus erroneously set down as dextrine. The percentages of cane-sugar are not strictly correct, because the presence of dextrine somewhat raises the results obtained, and there is no method, so far as I am aware, of making a satisfactory determination of cane-sugar and grape-sugar and dextrine in a mixture of the three bodies. But the method applied was the same in every instance, so that, besides the cane-sugar proper which is derived from the cereal used, the added percentage due to the error arising from the presence of dextrine would be nearly the same in every sample analyzed. The investigation of this problem should also come into the complete analyses of these foods, as well as the other matters spoken of hitherto. Scheibler, indeed, has indicated a method of detecting dextrine when mingled with cane-sugar in an amount not exceeding 3 per cent., but in these cases we have, as in Mellin's food, no less than 37 per cent. of dextrine, and 44.69 per cent. of grape-sugar as well, the cane-sugar itself being comparatively insignificant. His method is of no value for such cases.

CLASS III. MILK FOODS.

	15. Nestlé.	16. Anglo-Swiss.	17. Gerber's.	18. American Swiss.
Water	4.72	6.54	6.78	5.68
Fat	1.91	2.72	2.21	6.81
Grape-sugar and milk-sugar	6.02	23.29	6.06	5.78
Cane-sugar	32.93	21.40	30.50	36.43
Starch	40.10	34.55	38.48	30.85
Soluble carbohydrates	44.88	46.43	44.76	45.35
Albuminoids	8 23	10.26	9.56	10.54
Ash	1.59	1.20	1.21	1.21

These articles have been prepared in order to supply a food which should contain the constituents of milk to a certain extent, and yet should be free from the objections which condensed milk is open to. The attempt was first made by H. Nestlé in Vevey, Switzerland, but at the present time many milk factories are in existence, including one in our country at Little Falls, in New York, under the management of Dr. N. Gerber. All of these milk foods consist of cereals specially prepared in combination with milk. The preparation of the Anglo-Swiss milk food is stated to be as follows: 20 parts of Russian wheat flour and 20 parts of oat-meal are made into a dough and baked. The biscuit is then ground fine, mixed with 60 per cent. of condensed milk, dried by a slow heat at 120° to 130°, ground, and sufficient wheat gluten added to bring up the percentage of albuminoids to the same amount as that present in human milk. It is evident, that, apart from giving a general idea of the method of manufacture, this statement cannot be regarded as correct, inasmuch as the percentage of fat in the Anglo-Swiss milk food analyzed is much less than that which would be imparted by 60 per cent. of condensed milk. The percentage of albuminoids likewise makes it doubtful whether

any albuminoids in addition to those present in the milk and flour, have been added in the form of specially prepared wheat gluten.

According to Dr. N. Gerber (*Milk Analysis*, p. 70), the various milk foods in the market vary in composition as follows:—

		Average.
Water	5.0 to 10 per cent.	7.50
Salts	1.5 " 3 "	2.25
Fat	4.0 " 7 "	5.50
Albumen	9.5 " 18 "	13 25
Soluble carbohydrates	35.0 " 55 "	45.00
Insoluble "	15.0 " 25 "	25.00
Cellulose	0.5 " 1 "	0.75

It will be noted that Nestlé's food departs farther from the average than any of the other preparations, and the American Swiss approaches most nearly. The percentage of fat in the latter is much larger than in the other preparations, and the percentage of albuminoids is likewise the greatest. On preparing these various brands, the Nestlé, Anglo-Swiss, and Gerber's were very palatable and delicate in their flavor, more so than the American Swiss, which had a slight rancidity connected, no doubt, with the large percentage of fat and fatty acids. Under the microscope the various milk foods had a similar appearance, exhibiting agglomerations of starch granules and globules of milk. They all gave the starch and dextrine reaction with iodine, the reaction for dextrine being stronger in the Gerber than in the Anglo-Swiss. All had a faintly acid reaction except Nestlé's, which was slightly alkaline.

All of them have the same points in their favor, a high percentage of albuminoids, fats, and salts, this being especially true of the American Swiss. The conversion of the starchy matters into dextrine by

previous baking gives to this class of infant foods the advantages of that class of prepared cereal, which have been rendered easily assimilated by a process of previous torrefaction. The addition thereto of condensed milk has both advantages and disadvantages. The advantages are that the condensed milk is milk in a pure and safe form. Instead of being coagulated in large, cheesy masses in the child's stomach as would be liable to be the case if the condensed milk, after thinning with water, were given alone to the infant, the admixture of dextrine and torrefied milk keeps the caseine divided, and causes it to form in small flakes more nearly analogous to those forming from woman's milk. The condensed milk likewise adds a noteworthy percentage of fat, which is conspicuously absent from the other infant foods. It also adds a certain amount of milk-sugar and increases the percentage of albuminoids and valuable saline matters, more especially the phosphates. The principal disadvantage is, that condensed milk is preserved with the aid of cane-sugar, its analysis being as follows:—

Water	20.0	to 30.0	per cent.
Salt	1.5	" 3.0	"
Fat	8.8	" 12.0	"
Albuminoids	10.0	" 13.0	"
Milk-sugar	10.0	" 15.0	"
Cane-sugar	30.0	" 45.0	"

Cane-sugar, therefore, being relatively by far the largest constituent, there soon arrives a point in the manufacture of milk food when the addition of condensed milk must cease. Otherwise the percentage of cane-sugar, which like other carbohydrates is very objectionable when it takes the place of a proper amount of albuminoids, would become excessive, and

indigestion thereby be induced in the infant using such food. The remedy, it appears to me, would be found by using a condensed milk preserved without the aid of cane-sugar, and since this can now be successfully effected by means of Appert's method, the preparation of a milk food not open to the above objection should be soon satisfactorily accomplished. In that case we should have an infant's food with a very high percentage of albuminoids, fats, and salts; and a low percentage of carbohydrates. The sugar would be present in the form of milk-sugar derived from the milk, and as grape-sugar derived by a process of torrefaction from the meal. The last in its turn would not have to be present in larger amounts than what are requisite to supply the starch and dextrine, which are of use to prevent coagulation of the caseine in large masses.

CONDENSED AND PRESERVED MILK.

	Condensed (mean of 41).	Condensed (diluted).	Preserved Alpine.	Preserved Amer.-Swiss.	Preserved Amer.-Swiss. diluted.
Water .	30.34	88.39	58.57	59.21	87.78
Water .	12.10	2.02	13.21	11.55	3.46
Milk-sugar .	16.62	2.77	15.29	13.04	3.91
Cane-sugar .	22.26	37.1			
Albuminoids	16.07	2.68	11.36	14.10	4.23
Ash . .	2.61	0.43	1.78	2.09	0.62

It will be seen that when the condensed milk is diluted until its percentage of water is about the same as in human milk, it has relatively too little fat, and too little sugar (the larger portion of that also being cane-sugar) as compared with human milk. It has also too much albuminoids and ash.

Preserved milk is the result of scientific investigation applied to this subject, and its manufacture marks a great advance in the technique of the milk

industry. The product, when diluted, has no cane-sugar (an abnormal constituent of milk), and the ratio of its constituents is similar to ordinary cow's milk. It differs, of course, from human milk, and requires the addition of milk-sugar, fat, and of some bland inert attenuant, like torrefied wheat or barley flour, to overcome the general objection to any form of cow's milk as a substitute for woman's. Experiments in this direction are on foot, and will probably result in supplying a milk food not open to some of the objections urged against previous preparations of this class. The keeping qualities of the preserved milk are likewise very remarkable; its introduction is a great public boon, and cannot fail to exert a powerful influence upon the future of urban milk-supply, and the alimentation of infants.

Conclusions.—I have been frequently asked why I do not publish my own opinion as to the best of the various infant foods now in use. To do so would be very unwise for many reasons. But I have endeavored to do what I have regarded as of far more importance than this, which is to praise or blame just as the information afforded by physical and microscopic examinations and chemical analyses demanded, without partiality or bias, and to seek out and state the principles upon which, as it appeared to me, the dietetic value of these articles of infant food depended.

To summarize the points which I have endeavored to establish:—

1st. Cow's is in no sense a substitute for woman's milk.

2d. Attenuation with water alone is inadequate, and chemical metamorphosis, or, mechanically, the addition of some inert attenuant is required, in order to permit of the ready digestibility of cow's milk by infants.

3d. The utility of manufactured infant's food is to act as such attenuants, and as such they take the place of the simple barley and oat-meal water, the sugar, cream, baked cracker, arrowroot, etc. etc., used in former times.

4th. The results of both chemical and physiological analysis are opposed to any but a sparing use of preparations containing large percentages of starch.

5th. It is eminently probable that besides acting as attenuants, the matters extracted in the preparation of barley and oatmeal water, and still more the soluble albuminoid extractives obtained at ordinary temperatures (whereby coagulation is prevented), by Liebig's process, have a great independent value of their own. For this reason, instead of employing starch, gum, gelatine, sugar, etc., the use of a natural cereal extractive, containing saccharine and gummy matters and soluble albuminoids as well, such as our great and inspired teacher Liebig himself advocated, is in accordance with the developments of science since his time.

6th. The use of a food made up of equal parts of milk, cream, lime-water, and weak arrowroot water, as practised for years by the late Dr. J. Forsyth Meigs, and recently advocated by his son, Dr. Arthur V. Meigs, is sustained by theory, analysis, and practice. It provides for the increase of fat to an amount comparable to that contained in human milk. It adds

alkali to permanent reaction, and converts caseine into soluble albuminates. It adds a little bland attenuant. And if, in addition, the amount of milk-sugar were raised, and, instead of arrowroot water, barley or oatmeal water were substituted, as the case demanded, it would approach, it appears to me, still more nearly to the conditions required.

7th. The perfect solution of the present problem is to be found in the modification of cow's milk by chemical processes, so as to make it physiologically equivalent to human milk. The nature of these processes, and the results to be obtained, are at present so nearly wrought out, that there is good ground for believing that such a solution of this problem is not far distant in the future.

THE RELATIONS OF PAIN TO WEATHER, STUDIED DURING ELEVEN YEARS OF A CASE OF TRAUMATIC NEURALGIA.

By

Captain R. CATLIN,
U. S. Army.

WITH NOTES BY

S. WEIR MITCHELL, M.D.

[Read June 6, 1883.]

In *The American Journal of the Medical Sciences*, April, 1877, I described the case of Captain Catlin, U. S. Army, and gave the results of his observations on the relation of states of the weather to his own pain. These studies suggested by me, and carried out with care, at length became of equal interest to the observer—and, with occasional advice, he has continued to keep notes, until the record, with his comments, constitutes in my opinion a most valuable contribution to the strict science of medicine.

For full details of his case I must refer to my essay, but it is needful here to make a brief statement explanatory so that what he now says may be comprehensible without reference to the former record.

In August, 1864, Captain Catlin, then in perfect health, had his foot crushed in battle by a round shot. There was no shock; the leg was amputated below

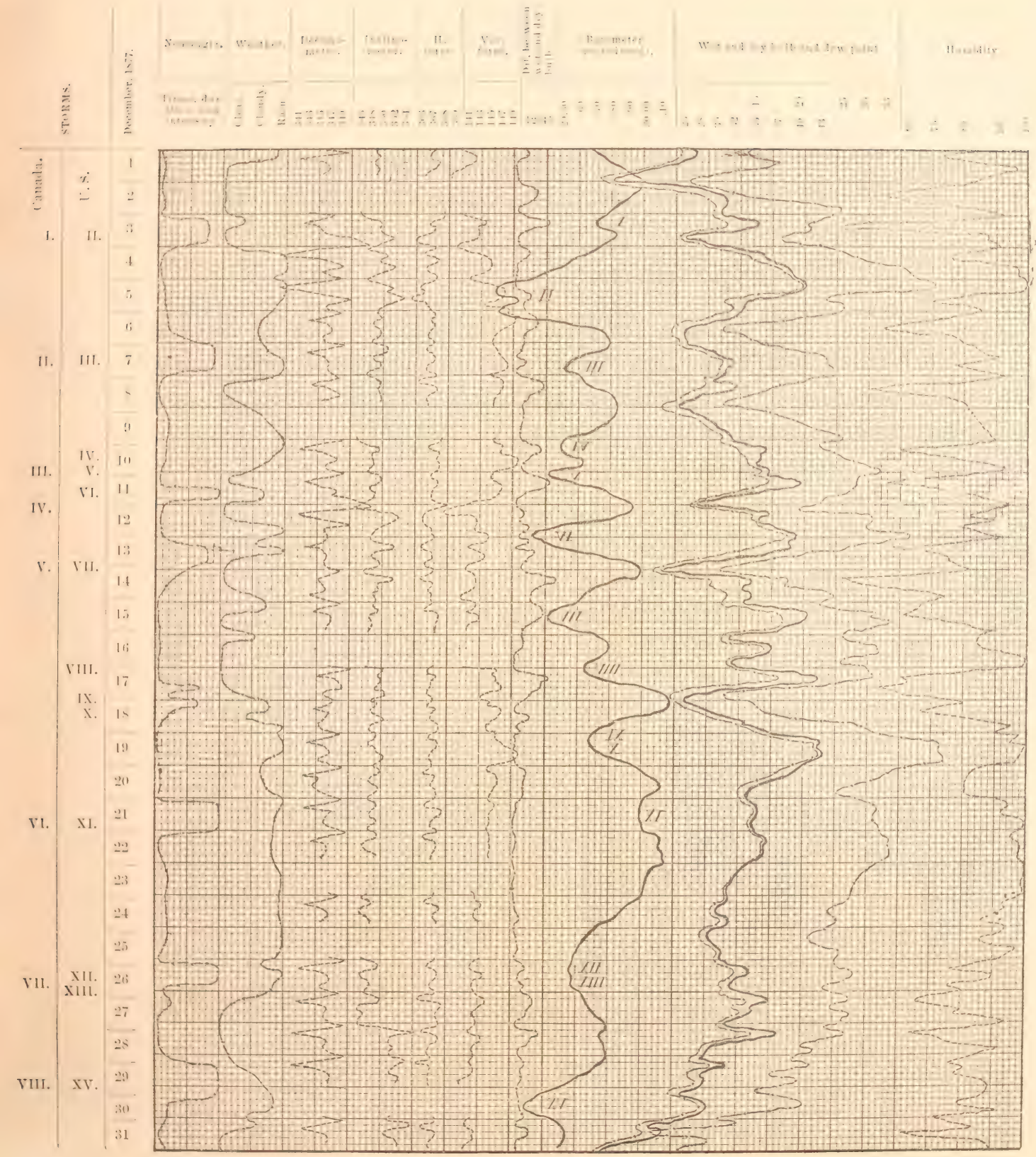
the knee, and he was up and about in a month. Pain was felt early in the lost foot, and became severe within nine months.

I should perhaps state that except as concerns pain the observer is in admirable health, that his attacks are so definite as to coming and going, as to create little difficulty in this direction, and that from his former position as instructor in certain scientific branches at West Point, he is well qualified by training to pursue this difficult study. The weather reports of the signal office and his own, or the daily observations of others, have enabled him, from time to time, to relate these to his record of neuralgia, and by this method he has avoided the constant presence of daily thought as to what was about to happen in consequence of atmospheric changes. I may add that I never knew any man more free from unwholesome attention to his own ailments. Most of what I learned from our former study, still stands unaltered by this much longer record; but there have been some fresh and suggestive gains in the relation of pain to meals, to daily barometric changes, and to magnetic conditions. The diagrams are readily understood in connection with explanations given in the text, except those for the months of November and December, 1877, which we have given as an illustration of the method of study, and of the obvious relation of storms to pain.

The following is the report of the case for the past seven years, made by Captain Catlin:—

For the year 1872 there were 1783 hours of pain (the records for '73 and '74 are incomplete); for 1875 there were

FIG. A.

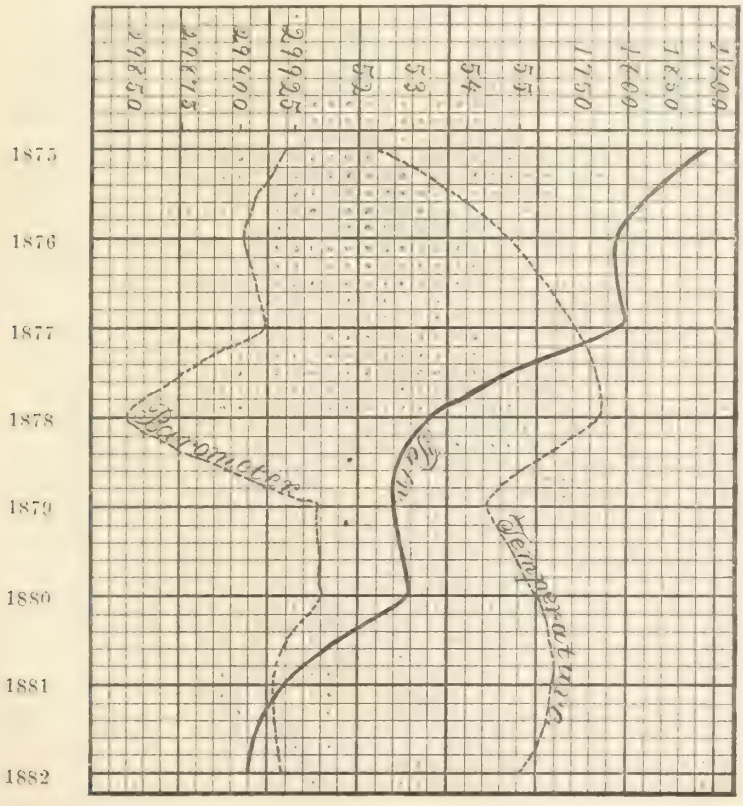

STORMS.
December, 1877.
Canada.
U. S.
I.
II.
III.
IV.
V.
VI.
VII.
VIII.
IX.
X.
XI.
XII.
XIII.
XV.
I
II
III
IV
V
VI
VII
VIII
IX
X
XI
XII
XIII
XV

1892 hours; for 1876, 1790 hours; for 1877, 1794 hours; for 1878, 1591 hours; for 1879, 1535 hours; for 1880, 1567 hours; for 1881, 1405 hours; and for 1882, 1370 hours. From 1872 the annual amount of pain increased and probably reached its maximum in 1874, a year of high pressure which as we proceed will be found to have some relation to the pain.

For 1875 we have the highest recorded amount of pain. Fig. C. The next year 1876, the amount of pain fell off 100

FIG. C.

Annual Pain, Barometer, and Temperature Curves, 1875–1882 *inclusive.*



hours, with a decrease of the mean annual pressure, equal to 00.021 of an inch, with corresponding increase of temperature. For the following year 1877, the pain and pressure remained nearly constant and parallel, with some increase in temperature. For the year 1878, there was a remarkable lessening of 200 hours in the neuralgic amount, but by following up the barometric curve an equally remarkable fall is found. The annual mean of the barometer for the year (1878) was 00.068 of an inch below the mean of the previous year, and 00.067 of an inch below the mean for the eight years: the mean for the eight years being 29.916.[1] From the low annual pressure of this year, we may almost infer a high annual temperature, which we do indeed find in the curve, and the Signal Bureau reported for the Middle Atlantic, the temperature "above the average" for every month in the year except June. For the year the *number* of pain attacks and the *number* of storms remained about normal, but the *duration* of the pain attacks was shortened.

This law of relationship of low pressure and high temperature to amount of pain, and the number of attacks of pain, and number of storms will become more apparent when we come to the consideration of the quarterly and monthly distribution of pain and storm. Since 1878 the pain has diminished at the rate of 40 hours annually; diminution being accelerated by a decrease of mean annual pressure and retarded (or actually held in suspension, as in 1880) by an increasing pressure. Making allowance for the annual decrease of pain, there is a remarkable parallelism between the pain and pressure curve. The yearly temperature bears the same relation to the barometer, as it was found to have in the monthly fluctuations. The total amount of pain for the eight years ending January 1st, 1883, was 12,944 hours, or nearly one-fifth of the time. The distribution of the pain is shown by Fig. D.—I., which is the curve

[1] For these barometric records I am under obligations to Prof. Daniel Draper, Director of the Observatory at Central Park, New York.

Neuralgic curve on monthly ordinates for 8 years, 1875-1882 inclusive.

Showing number of storms by the months for 8 years, 1875-1882 inclusive.

Mean monthly *duration* and *number* of neuralgic pains, 1875-1882 inclusive.

Monthly rain-fall for many years and for 8 years, 1875-1882 inclusive.

Monthly mean pressure for 8 years (New York), 1875-1882 inclusive.

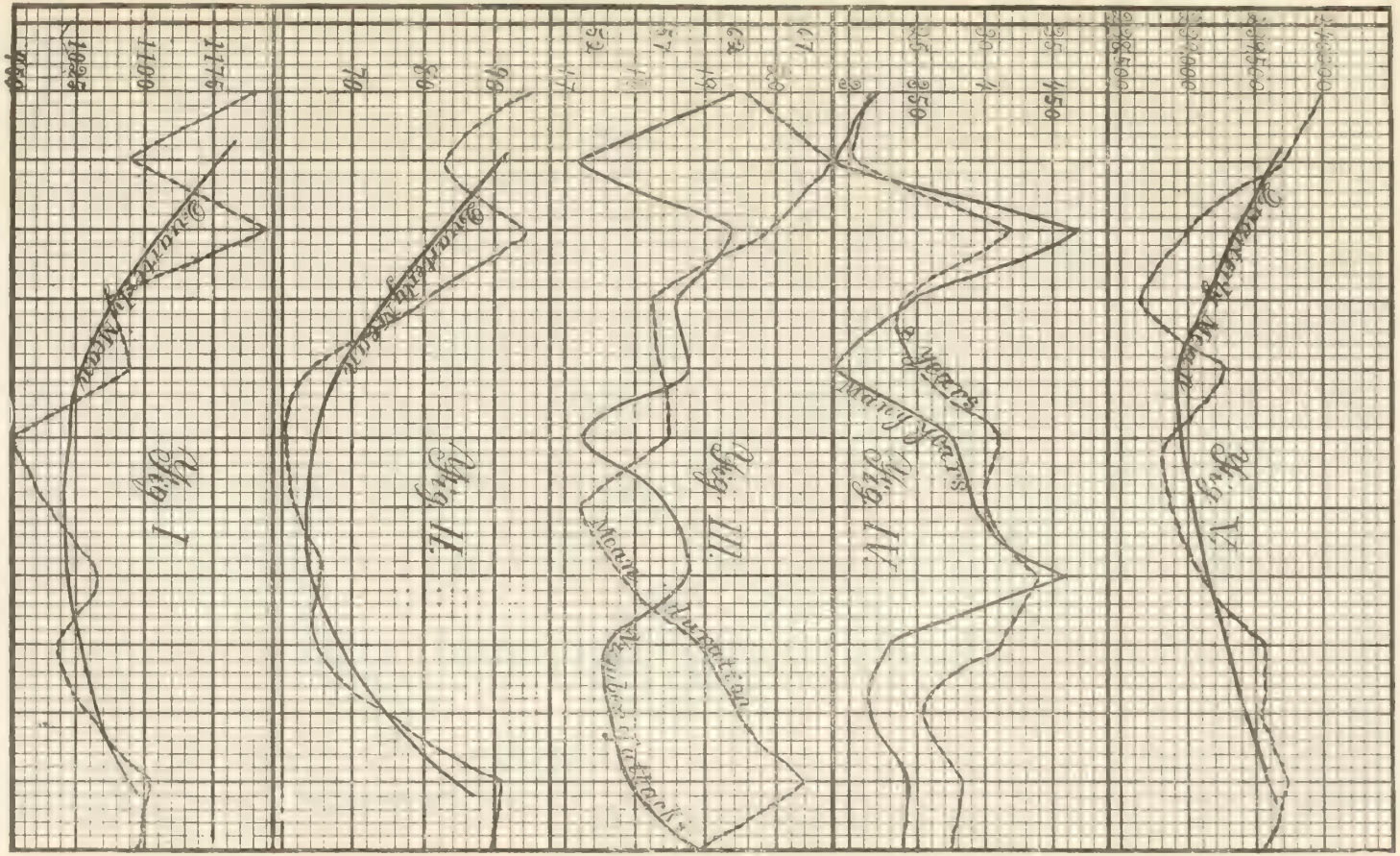


Fig. D.

of the total monthly amounts for the eight years. It will be readily seen that the winter months hold the advantage as pain-producers, and that for this period while the sun was north of the equator, there were 6783 hours, against 6161 hours while it was south of the equator.

Of the quarterly amounts, the first quarter, beginning with the winter solstice, leads with 3538 hours, followed by the fourth quarter at 3245 hours; then the second, with 3098, and the least in the third quarter (July, August, and September) with 3063 hours.

Of the monthly amounts, March holds the lead, closely pressed by January, and in order of amounts follow: November, December, May, February, April, August, October, September, July, and June. The number of storms, Fig. D.—II., recorded for the eight years is 806, taken from the Signal Bureau Records.

Of this number 529 belong to the winters, against 397 for the summers.

The quarterly numbers, in like manner, correspond to quarterly amounts of neuralgia, and by a reference to the storm curve Fig. D.—II., there is shown a remarkable parallelism throughout with the pain curve.

The average *duration*, Fig. D.—III., of each attack of pain for the whole time, was 18.97 hours; but again we find average *duration* bearing a relation to amount of pain and number of storms; the average for first quarter being 20.1 hours, for second quarter being 18.4 hours, for third quarter 17.9 hours, and for the fourth quarter 19.4 hours, which we see is directly proportional to storm and amount of pain. The average duration of pain was found to be greatest in February, 20.8 hours, one of the coldest, if not the coldest month, and containing probably the greatest barometric fluctuations of any month in the year.

Although the average velocity of translation of the winter storms be greater than the summer ones, owing to their much greater range and power they are further reaching,

and in consequence produce longer attacks. The hottest month of the year (July) gave the least average duration of pain, being seventeen hours.

This is a month of low average pressure and unimportant and minimum barometric fluctuations. By an examination of the curve of the mean monthly *number* of attacks, Fig. D.—III., there is found an almost exact *monthly* parallelism with the pain and storm curve, but the mean *quarterly* of the curve would not be as nearly parallel with the mean quarterly of the pain or storm, which shows that the number of attacks of pain is greater in proportion to the number of storms for summer than for winter. Many of the summer storms are difficult to trace, and some are not charted owing to their indefiniteness, but they are nevertheless pain-producers. For this reason it is believed that the relationship of the number of pain attacks to the number of storms is not as disproprtionate as appears in the quarterly comparison of the curves.

Passing to the rain-curve, Fig. D.—IV., we find it in harmony with the pain and storm curves, except for a few of the summer months. For these months, owing to the short and heavy precipitation of rain, and local character of many of the storms, the curve does not subordinate itself to the pain, storm, or even barometric curve.

Thus we see that rain-fall of itself is only relatively to be used as a measure for pain.

The barometric curve, Fig. D.—V., as it swings almost a catenary from January to December, measures with the storm curve much more accurately the ordinates of the pain curve ; and here we have reappearing in the quarterly and monthly products of neuralgia, the law already disclosed in the annual product of pain, viz.: high temperature, low barometer, favorable for non-product of pain; and in addition, minimum barometric undulations, favoring diminution of pain duration.

The following table gives the full record for years and months of the amount of pain in hours:—

TABLE OF MONTHLY AMOUNT OF PAIN 1875–1882 INCLUSIVE.

202 hours.	Jan.	1875	166 hours.	Jan.	1876	242 hours.	Jan.	1877
166 "	Feb.		175 "	Feb.		105 "	Feb.	
189 "	March,		188 "	March,		188 "	March,	
130 "	April,		173 "	April,		151 "	April,	
182 "	May,		158 "	May,		141 "	May,	
120 "	June,		116 "	June,		133 "	June,	
122 "	July,		111 "	July,		123 "	July,	
175 "	August,		132 "	August,		144 "	August,	
105 "	Sept.		155 "	Sept.		166 "	Sept.	
135 "	Oct.		105 "	Oct.		125 "	Oct.	
190 "	Nov.		160 "	Nov.		140 "	Nov.	
176 "	Dec.		151 "	Dec.		136 "	Dec.	
— 1892			— 1790			— 1794		

118 hours.	Jan.	1878	145 hours.	Jan.	1879	127 hours.	Jan.	1880
126 "	Feb.		131 "	Feb.		132 "	Feb.	
152 "	March,		140 "	March,		144 "	March,	
127 "	April,		121 "	April,		122 "	April,	
133 "	May,		116 "	May,		103 "	May,	
90 "	June,		150 "	June,		117 "	June,	
131 "	July,		132 "	July,		149 "	July,	
135 "	August,		133 "	August,		121 "	August,	
114 "	Sept.		122 "	Sept.		115 "	Sept.	
160 "	Oct.		121 "	Oct.		174 "	Oct.	
145 "	Nov.		115 "	Nov.		131 "	Nov.	
160 "	Dec.		159 "	Dec.		135 "	Dec.	
— 1591			— 1535			— 1567		

127 hours.	Jan.	1881	93 hours.	Jan.	1882
121 "	Feb.		128 "	Feb.	
127 "	March.		109 "	March.	
120 "	April,		118 "	April,	
144 "	May,		112 "	May,	
107 "	June,		114 "	June,	
98 "	July,		131 "	July,	
118 "	August,		102 "	August,	
105 "	Sept.		124 "	Sept.	
89 "	Oct.		129 "	Oct.	
123 "	Nov.		100 "	Nov.	
126 "	Dec.		110 "	Dec.	
— 1465			— 1370		

To determine the average distance of the storm-centre at the beginning of the pain attacks sixty well-defined storms through ten consecutive months were taken, and it was thus found that the average distance was 680 miles, ranging from 200 to 1200 miles.

Storms from the Pacific are felt the farthest off, very soon after crossing the Rocky Mountains. Those which move along the coast from the Gulf of Mexico, are associated with neuralgia not quite so intense, and are not felt, as a rule, until within the average neuralgia distance.

If neuralgia begins on a low and rising barometer, the ridge of high pressure between this and the coming storm-depression is narrow and invariably broken down within seventy-two hours, and more frequently within twenty-four or thirty-six hours; and during this rise, coincident with the pain, the difference between the wet and dry bulb thermometers, instead of increasing as is usual with this barometrical condition, sometimes actually diminishes, or increases for a few hours only, and then diminishes, showing increasing humidity.

When this pain and instrumental condition obtains, the coming storm depression will carry on its eastward side clouds and increasing moisture, and sometimes rain or snow, clear over the summit of the advancing high area pressure in front of it, holding an unusually high degree of relative humidity in the air on the high eastern slope of the high barometer area. These are the conditions when the pain attacks on the rising barometer. Its normal condition, however, is with a falling but it may be a high barometer, rising temperature, and increasing relative humidity.[1]

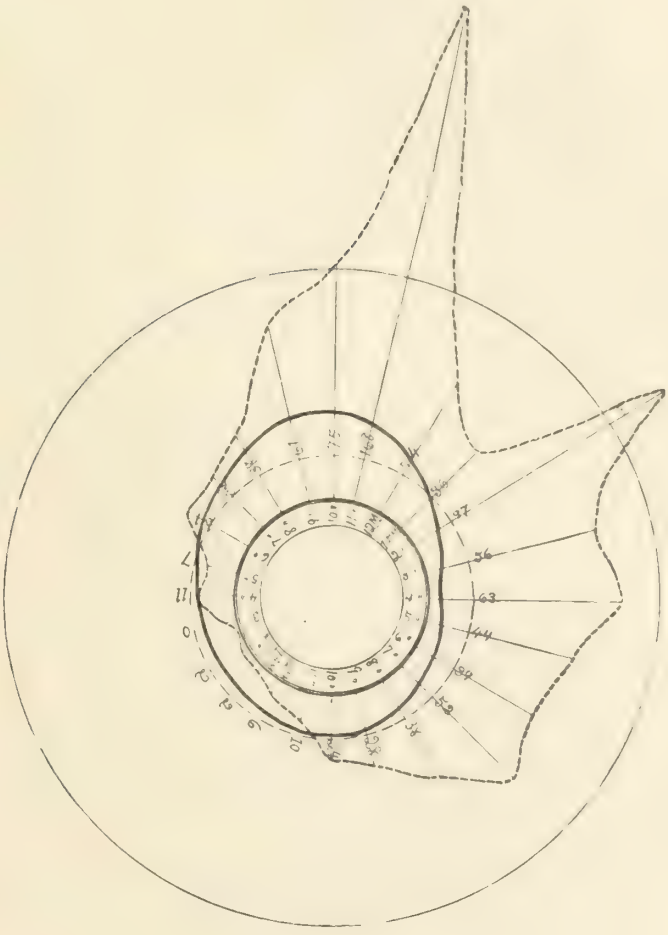
Should the pain be on during a day of intermitting rain, the pain takes an additional activity just before the increasing shower, and continues 20 to 40 minutes; this will sometimes happen four or five times in twelve hours. Each little increment of pain seems to bear about the same relation to the showers, as the main attack bears to the storm.

For the hourly relations of pain see curve Fig. E., which shows the number of times the pain began on each hour of the day. The time of record extends over a period of more than eleven years, commencing September, 1871, and is continuous since October, 1874.

It has always been observed that eating a meal, when the pain is on, intensifies it, and it is believed that it often hastens the attack. We should then expect to find some rise

[1] See same case in American Journal of Medical Sciences for April, 1877, p. 21.

Fig. E.



in the curve after each meal. This we do find at 9 A. M., which cannot be accounted for otherwise than that by eating at 8 A. M., some of the neuralgia, which really belongs to 10 o'clock, has been prematurely developed and so changed to 9 o'clock. Again after the 1 o'clock meal, there is a decided rise in the number, *some* of which, at least, should be chargeable to the meal. It is also worthy of note that there

is an ebb-tide in pain, just preceding the meals: not so marked before breakfast, partly, no doubt, because it is with me a little less regular than other meals, and partly because storms coming within neuralgia range during the early and middle sleeping hours, would not ordinarily arouse their victim, but would strike in the morning as the sleep became less profound.

This, doubtless, is the cause of the rapid rise in the curve, between 5 and 6 A. M.

In support of this statement of the respect which a storm may have for its neuralgia subject, it may be added, that if the pain is on when the patient falls asleep, and he is aroused or awakens during the night, he will find himself free from pain; but if he should move about, or stay awake 15 or 20 minutes, the pain is renewed, unless, in the mean time, the neuralgia time has expired.[1] At 11 P. M. the average number of attacks is not one a year, and from this time until 6 A. M., there is great immunity from *onsets* of pain.

With regard to the prominent and remarkable feature of the curve on the 11 A. M. ordinate, it cannot be positively stated why the pain has shown this great partiality; but there are some reasons which may be of interest to state. We have found that the years of high mean annual pressure gave high pain readings. We have also found that neuralgia in its relation to storms was not averse to a high barometer, but usually attacked on a falling barometer with rising temperature or increasing humidity.

The diurnal oscillation of the barometer is indicated by the elliptical curve in Fig. E., which has for its ordinates the distances from the curve to the dial circle, and for its abscissas the hours of the day. The difference between the

[1 This is true in regard to most pain, and may be within the experience of many men; we fall asleep, suffering, but awake without pain, even if the cause of pain remains. A brief period of being awake seems to be needed, in order that the cause become active. Sleep is a true anæsthetic.—S. W. M.]

maximum and minimum pressure is 00.045 of an inch. The minimum occurs about 4 P. M., and the maximum between 9 and 10 A. M., and begins its decided fall between 10 and 11. This brings the pain then in its relation to the diurnal change of the barometer to a position analogous to what we found it in its relationship to barometric movements accompanying storms—that is, high barometer, and on its fall preceding the storm, with increasing humidity and temperature. For the eight hours from 9 A. M. till 4 P. M., during the daily fall of pressure, there are 610 onsets against 346 for the remainder of the day, or 16 hours. The maximum amount of vapor is in the air from 2 until 6 P. M., increasing from before sunrise till just before sunset. We have then for the greatest daily neuralgic period a corresponding falling barometer, increasing vapor and temperature, with the analogous condition complete.

In regard to the 11 A. M. period, there are some other points of interest.

By a reference to the diurnal changes in the magnetic curves (Figs. A. and B.), we find: in the declination curve two maxima and two minima daily. The principal maximum occurs at 8 A. M., and the principal minimum at 2 P. M. From this minimum in its progressive movement there is a slight undulation in the curve about 10 P. M., giving rise to a subordinate maximum with another minimum following two hours later near the mean of the curve; from this we reach the principal maximum at 8 A. M.

In the inclination curve we have also two maxima and two minima. The leading and principal feature of this curve is its morning maximum which occurs between 10 and 12 M., probably 10.45 or 11 A. M. A subordinate maximum about 10 or 11 P. M. There is a minimum between 5 and 6 A. M., and another about 5 P. M.

Each of these maxima has a corresponding minimum in the horizontal force. So also the minima in inclination correspond in like manner to maxima in horizontal force. Owing to the great and precipitate change in declination

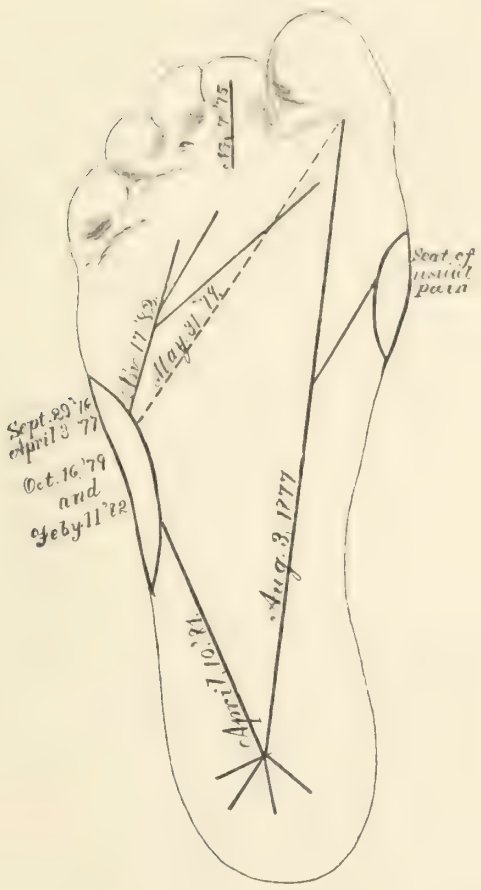
between 8 A. M. and 2 P. M. with the pronounced occurrences in inclination and horizontal force at or close to 11 A. M., may we not with some reason suspect these as active agencies in constructing the 11 A. M. neuralgic period?

The 2 o'clock period is one connected with the highest temperature and the maximum wind force of the day. It is believed that hours of maximum and minimum rain fall have some influence on this curve. The British Government[1] has published some observations which, although not conclusive, seem to show that there are two daily maximum rain falls, one at 7 P. M., and one at 5 A. M.; also two minimum periods, near noon and midnight. It is believed, further, from sensible impressions and limited observations, that solar and earth's radiation have an influence on the daily character of the neuralgia, and if fully studied would assist in interpreting some features of the curve not fully understood. With complete record of *local* meteorology, including radiation, there seems but little doubt that all the details of the diurnal curve could be demonstrated. There is one additional fact in connection with the onsets of pain which seems curious, and that is the period of drowsiness and sleepiness which precedes the attacks from one to five hours. This rule is not invariable, but is general.

For the seven years from Nov. 7, 1875, to Nov. 17, 1882, there have been nine neuralgic attacks of great and unusual power. These were characterized by pain in nerves not usually attacked. The ordinary neuralgia is confined to the position indicated in Fig. F., and is of the burning and boring kind, with twitchings of nerves in the stump, while in the extraordinary attacks, in addition to the pains as above named, we have the intense stabbing variety, with a much higher degree of the convulsive tendency. Accompanying each of these attacks except the last, we have the pain period dominated by more than one storm in six out of the nine attacks, and in *every* case the low barometric depression was of

[1] Prof. Loomis in Am. Jour. Sci. and Art, July, 1875.

FIG. F.



unusual and enormous extent. The attack of Nov. 7, 1875, began at 7 P. M. and lasted until 2 P. M. on the 8th, and was undoubtedly mainly under the influence of storm No. IX. (as charted by Signal Bureau), while passing from Savannah to a position off Cape May. The depression of this storm "became more extended as it advanced, with increasing velocity; the region of precipitation including the entire Southern and Middle States." By 7 P. M., closely following this attack, was an attack of the regular neuralgia, which

lasted 27 hours, to meet the requirements of Storm VI.; and this is a general rule, that these unusual attacks are followed by the regular neuralgia.

The attack of September 29 and 30, 1876, was under the influence of storms IX. and X. of that month. The weather report of the Signal Bureau says: "The map of the 29th, 4.35 P. M., shows that at that time a very large area of low pressure must have existed in British America on the south and east of James Bay, the southern end of which extended southward until on the morning of the 30th, the barometer was below 30 inches everywhere east of Lake Superior. The winds of Georgia and Indiana during this time were from east to southeast.

The attack of April 3, 1877, belongs to Signal Bureau Storms, Nos. II. and III. "An extensive area of cool northeasterly winds prevailed throughout the gulf States, Ohio Valley, and Lower Lakes." In Middle Atlantic States northerly winds moving to east with increasing cirrus and cirro-stratus cloudiness. The general depression extended over a large area. The attack of August 3, 1877, belongs to storm marked II. on monthly report. "There was a marked fall of the barometer in the Southern Atlantic States on the 2d, which district was then, in all probability, in the N. W. quadrant of an extensive depression." "N. E. winds prevailed from New York to Florida." The neuralgia attacked at 11 A. M., and at this time in the direction of the storm S. E. there were increasing cirrus and cirro-stratus with bunches of cumuli lower. This storm "united with No. I. over the St. Lawrence. Later in the day thunderstorms in northwest to northeast." The attack of May 31, 1879, corresponds to the extensive storm of No. XV. of Signal Bureau Report for the month.

The attack of October 16th corresponds to Storms IV. and V.; the former coming up the Mississippi Valley unites with the latter from the North Pacific, in Lake Michigan.

The attack of April 10, 1881, is related to Storm III.,

including Storms II. and IV. as described by the Signal Bureau Report.

It was observed on this occasion, as is frequently the case with this variety of pain, as also in the ordinary kind, that there was a cloudless sky with a delicate milky sheen in the atmosphere, more marked around the horizon. This milky veil becomes more opaque, and the first pronounced cloudiness will be small patches of cumulus, at lower elevation than this hazy appearance. These are storm indications, and favorable for the production of pain. This was the stabbing variety, as in each case here described, added to the other varieties, and was active between the heel and left side of foot. (See Diagram.)

The attack of February 11, 1882, corresponds to Storm VI. as marked in report of Signal Bureau. At the time the attack began, the centre of the storm was 600 miles westward. This was of wide lateral range, and is the only one charted for the month as coming from the Pacific coast.

For attack of November 17, 1882, Prof. Carpmail, Superintendent of the Toronto Observatory, reports more than ordinary declination and force disturbances from the 10th, including the 16th, when "they evidently showed signs of a coming storm." The magnetic storm set in at 5 A. M. on the 17th, and continued with great and intermitting force all day and night, and did not quiet down till midnight of the 18th. The report says, referring to the evening of the 17th, "that the force (horizontal and vertical) instruments, which in the early part of the storm showed a general decrease, now showed an increase, and between 7 P. M. and midnight, the lights several times travelled to both ends of the paper; the bifilar going off a number of times." The extremes of declination were reached easterly at 6.25 P. M., and westerly at 9 P. M. On the 19th, the magnets were disturbed. The disturbances being greatest between 11 P. M. of the 19th and 4 A. M. of the 20th. Many of the force movements were swift and rapid. The last movement of

importance occurring at 9.20 P. M. on the 20th. Brilliant auroras reported on the 17th.

This storm was unusually severe in England with brilliant aurora on the 17th. The disturbances here probably did not differ materially from those reported from the Toronto Observatory.

There was from the 10th instant, great unrest in all the nerves of the foot, such as feelings of twitching and slight burning, and at no time did the agitation touch the zero line of no pain. At 2 P. M. of the 17th the intense stabbing pains set in and reached the maximum power from 4 to 7 P. M. They continued, however, with great force until 5 A. M. on the 18th, and intermitting and less strong fits of torment were felt till evening; then a period of rest occurred until 9 A. M. of the 19th, when a severe attack of the regular neuralgia set in, with more or less burning and twitching pains throughout the foot, which lasted more than thirty-one hours, and finally went off on the evening of the 20th. The intense neuralgia of the 17th seems connected with the magnetic storm of the 17th, for there was no storm of barometric depression charted by the Signal Bureau within neuralgic range for this date. The last storm preceding this time was storm No. II., which started from the Pacific on the 8th, and for which there was a regular neuralgia on the 9th and 10th. The next storm was No. III., which originated in Texas on the 18th, and the attack of pain of the 19th and 20th was for this storm aggravated by the magnetic condition of these dates. It is firmly believed that neuralgia accompanies intense auroral periods, but owing to their rare occurrence, it cannot be said that the proof is conclusive. The conjunction of the two seems too frequent for mere coincidence.

In regard to treatment, etc., I can say but little. There has been no treatment in a medical way of late. I have had good health, take a great deal of exercise, but in a rather irregular way; my appetite is always good, and I sleep well, except when the disturbance of neuralgia interferes. For the

year 1878, in which occurred the greatest diminution of pain for any year, there was the greatest physical weight, reaching 190 pounds. Physical exercise, nutritious food (have found milk most fattening of all), and light, agreeable occupation, are, I found, the best regimen for this neuralgic subject.

A CLINICAL STUDY OF THE CRANIAL NERVES.

By

HARRISON ALLEN, M.D.,

PROFESSOR OF PHYSIOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

[Read June 6, 1883.]

THE cranial nerves at their nuclei of origin and in the course of their intrinsic fibres may be involved in diseased action. It would seem to be more than a coincidence that the nuclei of the ninth, tenth, and eleventh nerves are arranged in a definite series, forming the trineural fasciculus; and that the nerves themselves are associated in function. But this line of reflection is useless when applied to the nuclei of the sixth and seventh nerves. These nerves appear to have no function in common, and yet they arise, in part at least, from the same nucleus. In their range of variation they have no relation with one another; nor in any animal known to the writer does the sixth nerve spring from the facial trunk, but always (in variations from the human type) from the third. The third, fourth, and sixth nerves are naturally associated; but no reason is assigned for the withdrawal of the nucleus of the sixth from the mid-brain, where the nuclei of the third and fourth nerves lie, to the hind-brain, where it is in association with an alien, namely, the seventh. Associated paralysis of the

third, fourth, and sixth nerves is common; but paralysis of the sixth and seventh is rare.

The different symptoms of encranial paralysis may be assigned to one of two groups of causes, viz., to pressure, or to injury. Pressure may affect a nerve remote from the origin of the pressure. A tumor of the cerebellum, for example, has been found to be associated with a facial palsy, although neither the nuclei nor the intrinsic fibres of the seventh were involved. Clinical reports do not discriminate between the direct effects of injury and the indirect effects of pressure. Until the influence of this pressure is known and in each case eliminated, the study of localization of diseased action in the brain must remain in an unsatisfactory state.

Among the localities in which the effects of local pressure have been determined may be mentioned the pontal portion of the medulla oblongata, at which place the inferior vermiform process of the cerebellum may press against the nuclei of the cranial nerves there situated; the third ventricle, where from a tumor in the corpora quadrigemina, or the pineal body, the lymph of the lateral and third ventricles accumulates in these chambers and presses downward, notably at the floor of the third ventricle and the lamina cinerea; the region of the hippocampus in the descending horn of the lateral ventricle, which may be distended by the accumulation of fluid in senile states of the brain. Even where the fluid from the larger ventricles is conceived to pass freely along the iter to the fourth ventricle, it is fair to suppose that if this fluid is excessive in quantity, undue pressure will be exerted on the walls of the iter, and that

some instances of paralysis of the third and fourth nerves might have their origin in this way.

The causes due to injury of the nerves are of two kinds; primary, when the lesion is due to the direct pressure of a tumor, or a clot of blood; and secondary, when it is caused by the deteriorating influence of a tract of degeneration in the neighborhood.

Of the first-mentioned, that is to say, of the effects of primary pressure, there is abundant clinical evidence; indeed, it is the only evidence which is indubitable. Of the second, it must be acknowledged that the evidence is purely hypothetical; and as far as the writer knows no case can be adduced in its support. It may be more than a coincidence that the study of frontal (transverse) sections of the medulla and of the stem of the brain shows that the nerves most commonly paralyzed in hemiplegia of encephalic origin are those whose intrinsic fibres lie in the immediate neighborhood of the pyramidal tract, as this has been defined by Türck, Flechsig, and Charcot. Thus the third, sixth, seventh, and twelfth nerves would be easily influenced by diffusion from the tract of degeneration, while the remaining nerves of the series would escape.

[After the reading of the preceding paper:—]

Dr. Charles K. Mills said: That the subject touched upon by Dr. Allen was one of great interest to him. With reference to the effects of pressure upon the nuclei of the cranial nerves, he said that, without doubt, some extraordinary and apparently inexplicable affections of cranial nerves were due to such pressure. Intraventricular effusion, however, such as occurs in hydrocephalus, was not likely to produce local paralysis or spasm, because, owing to the free communication between the various ventricles, unless such effusion took place suddenly, the nerve-centres and conductors seemed to accommodate themselves to the pressure, which was distributed over a large area and over nearly all districts alike.

Those cranial nerves which had the greatest peripheral exposure, and the longest and most tortuous course in bone and membrane (the facial, oculo-motor, etc.) were most liable to be affected by syphilitic, rheumatic, gouty, and other similar diseases.

He supposed that the facial and abducens were so often affected independently, because paralyses of these nerves were usually peripheral; the lesions causing them being situated not only beyond the nuclei, but commonly beyond the superficial origins of these nerves. Besides, some authorities claimed separate, although near, nuclei for the sixth and seventh. He had, however, seen more than one case of conjoint paralysis of the abducens and portio dura, presumably due to degeneration of the combined nuclei.

He recalled one case, which he did not think had been put on record, but was well worthy of being detailed in connection with a study of these affections of cranial nuclei. The patient was sent from the South to the Hospital of the University of Pennsylvania by Dr. John Guitéras, and was the victim of complete double oculo-motor paralysis. Associated with this was advanced atrophy and paralysis of both upper extremities. Here he believed the lesion to have been bilateral degeneration of the nuclei of the cervico-brachial nerves and also of the oculo-motor nuclei. One of the strange features of the case was the fact that the cranial nuclei between oculo-motor and cervical regions (those of the pathetic, trigeminal, facial, abducens, glosso-pharyngeal, etc.) had escaped.

Dr. Mills said, also, that the present was perhaps a good opportunity to put on record an observation on cerebral glosso-labio-pharyngeal paralysis. It is well known that this affection is usually attributed to degeneration of the cranial nuclei; in fact, it is often called "bulbar paralysis." A patient presenting the oral, labial, lingual, and pharyngeal symptoms of this disorder, had died about two years before at the Philadelphia Hospital. Examination showed no lesion of pons or medulla. Along the outer border of both lenticular nuclei, and extending into the external capsule and claustrum, were long, narrow, and somewhat irregular areas of degeneration; softened or broken-down districts surrounded by walls of harder, darker tissue. Kirchoff and others had since reported similar cases, but up to the time of making the observation he had never seen such a case reported.

In conclusion, he said that Dr. Allen had discussed the probability of lateral diffusion of descending degeneration. He thought this lateral extension of degeneration more likely to occur at the top of the system than in the motor tracts, from cell to cell in the central zones rather than from fibre to fibre in the conducting columns. He confirmed Dr. Allen's remarks with reference to the practicability of naked-eye observations of degenerated areas and tracts; and spoke of the brain of a hemiplegic negro murderer, presented by him to the Philadelphia Pathological Society, which showed atrophy of the right cerebral and of the left cerebellar hemisphere, and a connecting tract of degeneration through the pons.

Dr. W. W. Keen referred to one point mentioned by Dr. Allen, the question of the common nucleus for the sixth and seventh nerves; and called attention to an extremely rare and interesting

Case of Congenital and Bilateral Paralysis of the Sixth and Seventh Nerves.—This case he had been requested to see by Dr. Geo. C. Harlan on account of its bearing on the artistic anatomy of the face. Dr. Harlan had reported it in full in the *Trans. Amer. Ophthal. Soc.* for 1881. Dr. Keen had had his face photographed as well as copies taken of a photograph in childhood. Unfortunately the existence of the latter was not known until the more recent photograph had been taken, or the point of view would not have been reversed. Copies of the photographs, Figs. 1 and 2, are appended, as they do not appear in Dr. Harlan's account of the case. Fig. 1 is a fairly good reproduction, but Fig. 2 is poor.

FIG. 1.

FIG. 2.

At the date of examination he was 18 years old. His expression in the second photograph it will be seen had scarcely changed at all from that of the earlier one. His face had simply enlarged. The condition had existed from birth. Not a muscle of the face responded either to the will or to the battery. The corner of the mouth could be slightly depressed by the platysma. Salivation was normal; hearing was good; speech perfectly intelligible, and only affected by the want of control over the lips; taste and sensation in the tongue were unaffected.

There was double internal strabismus; the cornea was slightly dull; vision O. D. $\frac{20}{126}$ and O. S. $\frac{20}{c}$; not improved by glasses; the impairment was chiefly due, apparently, to constant epiphora; ophthalmoscopic examination unsatisfactory, but the fundus seemed normal. Fixation was with the macula in each eye. No diplopia.

The absolute, bilateral and congenital absence of function of the sixth and seventh nerves, without any involvement of the chorda tympani, points clearly to defective development of the nucleus common to the sixth and seventh nerve, and tends to confirm the assertion of Sapolini (*Amer. Journ. Otology*, Oct. 1881, p. 312) of the differentiation of the chorda by origin from a distinct nucleus as a thirteenth cranial nerve.

In reply to the discussion elicited by his remarks, Dr. ALLEN thought that the manner in which the effects of locomotor ataxia are witnessed in the spinal cord, confirmed his own impressions with respect to the manner by which diffusion can take place about a tract of degeneration. It is well known that in this disease the central ravages are not confined to the postero-lateral column in which they are initiated, but extend to the postero-median column, and may even pass over the postero-median fissure. A yet more striking instance is seen in the experiment of Gudden, who extirpated the eyes of rabbits and observed the change that took place in the optic tracts. It was found that the paths of degeneration were not limited to the visual lobes of the corpora quadrigemina, but had involved adjacent structures by diffusion. The effects of degeneration seen in the peripheral nerve-trunks, where they were strictly limited by reason of the medullary sheaths, would not serve as precedents for the localization of similar effects in the central nervous system where such sheaths are so often absent.

SOME OBSERVATIONS ON THE SALIVARY DIGESTION OF STARCH BY INFANTS.

By

J. M. KEATING, M.D.,

VISITING OBSTETRICIAN TO THE PHILADELPHIA HOSPITAL, AND LECTURER ON DISEASES OF WOMEN AND CHILDREN.

[Read June 6, 1883.]

Recently, in a late English work, I find the following: "during the first few months farinaceous food of every kind should be avoided, for the child's stomach (?) cannot digest it. Until the third month, or even later, no saliva is secreted, and without this floury foods cannot be assimilated." (*Management of Infants, etc.*, by Howard Barrett.) This idea is so prevalent, and most of us have adopted the statement as representing the teaching of physiologists, that it has always been a matter of surprise to those interested in the feeding of infants to find occasionally, especially amongst the poorer classes, infants fed upon corn-starch or other farina, almost to the exclusion of other food, and thrive.

At present, we presume that amylaceous material has of necessity to be converted by hydration into glucose, and for this reason I will not detain you this evening by indulging in the more speculative aspect of this subject, as to whether dextrine is capable of being absorbed, and which of the two ferments, that of the salivary glands or of the pancreas, is of the

most importance. We have left this matter for further investigation. Prof. Albert R. Leeds, in his very able exposé of the subject of foods as regards their chemical constituents, made the matter so clear, that in his table of the analyses we have evidently an accurate guide for the selection of foods in individual cases. But as the general belief is, that for infants of a tender age, our choice should fall on that which contains a minimum quantity of starch and a maximum amount of vegetable albuminoids, or foods based on the Liebig formula, I deemed it valuable to institute a series of experiments, the results of which I confess were rather surprising, to test the saliva of infants, and to satisfy myself that the reason why some children apparently thrive on starchy food, is not due to any change in the starch in its preparation, but depended upon contact with secretions well established in childhood.

In these tests we endeavored, as far as possible, to exclude all error. Corn-starch was used, it having been previously boiled, cooled into a paste, and portions of this were put into little linen bags, and given to infants to suck for two minutes at a time; Pavy's test was then used; the corn-starch paste exhibited before the experiment no evidence of sugar change.

The linen was thoroughly boiled without discoloration of the solution. The bags with their contents were in each case thrown into a test-tube, and I submit for your examination the accompanying report.

To the resident physicians of the Philadelphia Hospital, Drs. B. F. Hawley and A. E. Roussel, I am indebted for assistance in this matter, as many of these tests were repeated a number of times by them,

and great care was used to insure accuracy. My report includes the results obtained by experiments with the saliva of twenty-one children—varying in age from six days to seventeen months. The sugar-change was noted in all but three—one of these was a babe six days old—whilst in another babe seven days old a marked reaction was observed. I feel satisfied that some infants do digest starch provided it is presented to them in a digestible form, and also that the salivary secretion, which occurs earlier than we have been accustomed to believe, is allowed to come in contact with it; and I cannot but attribute the many statements to the effect that starchy food in small quantities is contraindicated, to the fact that the secretions of the mouth are less liable to exert their influence when such food is administered by bottle and deposited in a surprisingly short time in the acid juices of the stomach. If starchy food, such as barley flour, oatmeal, rice, wheat flour, etc., is indicated on account of its highly nutritious qualities which exist in all portions of the grain, and deemed advisable in the feeding of children, our few observations teach us that they should be administered in such a way as to insure their thorough digestion; and I am satisfied that the surprising results we witness, especially among the poor, of thriving table-fed babes, is due to the mode of feeding more than to the fact that they are exceptional and astonishing cases. My table shows that the age of the infant is not a guide to the quality of its saliva, and we should bear this in mind when choosing the form of food. Thus, should we be called upon to regulate an infant's feeding, it would be important for us to test the saliva.

If we find a sugar change taking place, we might incorporate with milk small quantities of one of the cereals, either barley, oatmeal, or wheat. On the contrary, should the test prove negative, Horlick's or Mellin's food would be decidedly preferable. But while thoroughly convinced that the saliva is a most important element in digestion, we cannot overlook the fact that starchy foods have also to run the gauntlet of the pancreas, which organ, if it possess in childhood relatively the same power that it does in later years, is far more active than the salivary glands. We cannot then overlook the value of a microscopic examination of the stools of all bottle-fed children, for I believe that by this alone we can regulate the quantity of farinaceous food, detecting the proportion of the undigested residue.

Child's name.	Age.	No. of teeth.	Food.	Reaction.	No. of experiments.
Smith,	6 days	0	Breast.	None.	3
Coyle,	7 "	0	Breast.	Marked.	1
Gallagar,	11 "	0	Breast.	Well marked.	2
Asbulson,	12 "	0	Breast.	Marked.	1
Perry,	2 weeks	0	Breast.		1
McErwin,	3 "	0	Breast and crackers.	Well marked.	3
Meenan,	3 "	0	Bottle.	Perceptible.	2
Conner,	4 "	0	Breast.	Marked.	2
Davis,	4 "	0	Breast.	Marked.	1
Beatty,	4 "	0	Breast.	Very slight.	2
Sumley,	4 "	0	Breast.	Marked.	1
McCann,	4 "	0	Breast.	Very marked.	1
Newhouse,	7 "	0	Breast.	Very slight.	2
Roberts,	2 mos.	1	Breast.	Marked.	2
Nerain,	2 "	0	Breast.	Very marked.	1
Boenning,	2 "	0	Breast.	Marked.	1
Hemileth,	4 "	2	Corn-starch and crackers.	Well marked.	2
Roach,	5 "	1	Corn-starch and crackers.	Slight.	3
Hall,	8 "	2	Breast and crackers.	Marked.	1
Devine,	13 "	4	Corn-starch and crackers.	Well marked.	1
Wood,	17 "		Condensed milk.	None.	

The following are my conclusions:—

The saliva of some infants possesses the property of converting starch into glucose regardless of age.

The age of the infant cannot be taken as an indication of this property of its saliva.

When such a condition is found to exist, a small quantity of well-prepared farinaceous food is valuable as an element in the diet, incorporated with mixed cow's milk.

An examination of the stools of children so fed, would be a guide as to the quantity of starchy food to be used, and when farinaceous food is employed slow feeding is probably preferable to the bottle.

A NOTE ON THE FECES OF STARCH-FED INFANTS.

By

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ASSISTANT DEMONSTRATOR OF PHYSIOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

[Read June 6, 1883.]

The series of experiments presented in the preceding paper by Dr. Keating seems to me to be in the highest degree suggestive, for it is only rational to suppose that the development of the amylolytic ferment of the pancreatic juice is coincident with the appearance of the analogous salivary ferment. Inasmuch, however, as the food even in spoon-fed infants is retained but a short time in the mouth, and further, as the continued action of the saliva after it enters the stomach is as yet problematical, the only absolute control for such observations is afforded by an examination of the feces.

Through the kindness of Dr. Keating I have been enabled to examine the stools of twenty-four starch-fed infants, of ages varying from forty-five days to eighteen months. Twenty-three of these children were fed upon cracker-dust, water, and condensed milk. The twenty-fourth received corn-starch boiled in milk.

The freshly evacuated feces of each infant were

carefully bottled and labelled, and a drop of a solution of iodine was added to a small portion of each specimen, which was then submitted to microscopical examination. Besides turning the starch blue, a ' indicating the presence of dextrine by a peculiar m. hogany-red color, the iodine has the advantage o. rendering any fats which may be present much more readily apparent. The reaction of each specimen was taken, but though this varied from acid to alkaline and neutral, no correlation between the reactions and the other properties of the specimens could be observed. A decoction of each was tested for glucose with freshly prepared Fehling's solution, but except in one instance no appreciable amount could be found.

The presence of *starch* was exceptional and apparently in no degree dependent upon the age of the child. The stools of eighteen out of the twenty-four children contained either no starch, or but a trace, *i. e.*, no more than is frequent in the evacuations of a healthy adult upon a mixed diet. Six of these specimens were from children of three months or less,—the youngest being but forty-five days old. In many cases the broken and empty cellulose envelopes of the starch granules were clearly discernible.

The six infants in whose evacuations a noteworthy amount of starch was present were aged respectively three, four, ten, thirteen, fourteen, and seventeen months. The eldest two were in very bad health.

The following is a tabular statement of the age, diet, and appearances of the feces in the children forming the subjects of this study.

AN EXAMINATION OF THE FECES OF TWENTY-FOUR STARCH-FED INFANTS.

No.	Name.	Age.	Food.	Starch present.	Remarks.
1	Savin,	45 days	Condensed milk and cracker dust.	None	
2	Jocker,	2 mos.	"	Traces	
3	McGettinger	2+ "	"	"	
4	McGowan,	3 "	"	"	Twice examined: no fat before inunction, about 10 per cent. after.
5	Ross,	3 "	"	"	
6	Hays,	3 "	"	About ¼ starch	
7	Soy,	3 "	"	Traces	
8	Henwich,	4 "	Corn-starch and milk.	"	
9	Moore,	4 "	Condensed mi'k and cracker dust.	None	Many broken cellulose envelopes.
10	Conway,	4+ "	"	Traces	Evidences of potato surreptitiously given
11	Roach,	5 "	"	About ¼ starch	
12	Anxier,	5+ "	"	None	
13	Schmitz,	5+ "	"	"	Many bacteria.
14	McKinley,	6+ "	"	"	10 per cent. fat; had had inunctions.
15	Hall,	8+ "	Breast and cracker food.	Traces	
16	Hensen,	10+ "	Condensed milk and cracker dust.	More than normal	Many bacteria; evidences of potato surreptitiously given.
17	Devine,	13— "	"	20 to 30 per ct.	Some glucose present and indications of dextrine; saliva was found to be inefficient.
18	Croncia,	14— "	"	Traces	
19	Madden,	14 "	"	"	
20	Boyle,	14 "	"	10 p.ct. starch	Sick.
21	Glass,	14+ "	"	None	Except a few large cells containing starch from potato.
22	Kinscher,	17— "	"	"	
23	Wood,	17— "	"	Over ¼ starch	Syphilitic; saliva was found to be inefficient.
24	Dane,	18 "	"	Traces	Indications of dextrine.

The facts presented appear to justify the following conclusions:—

First, that *many* infants of under three months can digest starchy foods;

Second, that the individual variations in this regard

are so numerous that no broad and general statement can be made as to the period at which infants *begin* to digest starches; and

Third, that the physician can be absolutely certain that a farinaceous ingredient in the diet of a young infant is beneficial only by an examination of the dejecta under such diet.

[After the reading of the preceding paper:—]

Dr. KEATING spoke of one case in which fat was found in the feces—cod-liver oil had been administered in the form of inunctions, and the child fed alone on Borden's condensed milk.

Dr. RANDOLPH replied that he had found 10 per cent. of fat in the feces of a child which was receiving two inunctions of cod-liver oil daily. He was now conducting some experiments upon this subject, and intended to report the results to the College in a communication to be read at a future day.

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